THE UNITED STATES DISTRICT COURT
FOR THE MIDDLE DISTRICT OF PENNSYLVANIA

IMEN GHARBI and HATTAB (BEN) GHARBI, as Co-Administrators of the Estate of E.G., deceased and IMEN GHARBI and HATTAB (BEN) GHARBI, Individually and in their own right,	:	CIVIL ACTION NO. 1:19-CV-1943
Plaintiffs,	:	(JUDGE MARIANI)
v.	:	
THE UNITED STATES,	:	
Defendant.	:	

MEMORANDUM OPINION

I. INTRODUCTION

The Plaintiffs in this matter are Imen Gharbi and Hattab (Ben) Gharbi, the parents of deceased E.G. Mrs. Gharbi was admitted to Harrisburg Pinnacle Hospital on December 16, 2017, for induction of labor. Potatia Francis, M.D., assumed Mrs. Gharbi's care at approximately 8:00 a.m. on December 17, 2017, and delivered E.G. by cesarean section at approximately 1:41 a.m. on December 18, 2017. After resuscitation efforts failed, E.G. was pronounced dead at 2:00 a.m. on December 18, 2017.

Because Dr. Francis was employed by a federally funded health clinic at the relevant time, the above-captioned action for medical malpractice is brought against the United States under the Federal Tort Claims Act ("FTCA"), 28 U.S.C. §§ 2671 et. seq. Plaintiffs maintain that

> Dr. Francis failed to meet the standard of care with respect to establishing a plan of care for delivery, actively and closely monitoring and managing Mrs. Gharbi's labor, recognizing arrest of active labor necessitating cesarean delivery, and recognizing the 14-minute deceleration and sentinel bradycardic Category III fetal strip as necessitating urgent cesarean delivery, and that as a result of Dr. Francis' failure to timely deliver, E.G. died. E.G.'s death was foreseeable and preventable. To a reasonable degree of medical certainty, E.G.'s death was as a direct and proximate result of critically decreased oxygen in utero with hypoxia-induced decelerations and absent to minimal fetal heart rate variability due to protracted induction and labor and failure to timely deliver.

(Doc. 116 at 2.) Defendant asserts that E.G.'s death was not caused by anyone's negligence. (Doc. 117 at 3.)

In a six- day bench trial which began on February 22, 2022, and ended on March 2, 2022, the Court heard testimony from the following witnesses:

1. Potatia Francis, M.D.- attending Obstetrician and Gynecologist (Trial Transcript ("TT") Day 1 (Doc. 108), 18:16-18);
2. Jill Mauldin, M.D. - offered by Plaintiffs and accepted as an expert in Obstetrics and Gynecology, Maternal Fetal Medicine, and Electronic Fetal Monitoring (TT Day 1, 137:24-138:4);
3. Jennifer Hammers, D.O. - offered by Plaintiffs and accepted as an expert in Clinical, Anatomic, and Forensic Pathology (TT Day 2 (Doc. 109), 12:7-12);
4. Royal Bunin - offered by Plaintiffs and accepted as an Actuarial and Economic Expert (TT Day 2, 108:4-7);
5. Plaintiff Hattab (Ben) Gharbi (TT Day 2, 141);
6. Plaintiff Imen Gharbi (TT Day 2, 173);

7. Chad Staller - offered by Defendant and accepted as an expert in Forensic Economics (TT Day 3 (Doc. 110), 6:19-24);
8. Brooke Crummel - UPMC Pinnacle Hospital Respiratory Department System Blood Gas Manager (TT Day 3, 36:15-37:3);
9. Wendy Chung, M.D. - offered by Defendant and accepted as an expert in Medical Genetics and Clinical Pediatric Genetics (TT Day 3, 57:25-58:1));
10. Christian Pettker, M.D. - offered by Defendant and accepted as an expert in Maternal Fetal Medicine (TT Day 4 (Doc. 111), 11:25-12:1);
11. Toni Heubscher Golen, M.D. - offered by Defendant and accepted as an expert in Obstetrics and Gynecology (TT Day 5 (Doc. 112), 112 8:6-7, 10:5-7); and
12. Richard Polin, M.D. - offered by Defendant and accepted as an expert in Neonatology and Pediatrics (TT Day 6 (Doc. 113), 8:24-25).

In addition, the Deposition Transcripts of Gerald F. Maenner, M.D., and Megan Klamerus, D.O., were read into the record. (TT Day 1, 127:16-17.) Dr. Maenner was Mrs. Gharbi's prenatal obstetrician and the attending in charge of her care from the time of her admission for induction on December 16, 2017, until 8:00 a.m. on December 17, 2017, when Dr. Francis took over as the attending obstetrician. Dr. Klamerus was the resident working with Dr. Maenner on December 16th and 17th.

The Court must resolve the following issues:

1. Whether Plaintiffs have shown by a preponderance of the evidence that Dr. Francis failed to meet the standard of care in her handling of Mrs. Gharbi's labor and delivery;
2. If Dr. Francis failed to meet the standard of care, whether Plaintiffs have shown by a preponderance of the evidence that the breach of the standard of care was the factual cause of the death of E.G.; and
3. If Dr. Francis's failure to meet the standard of care was the factual cause of the death of E.G., what damages resulted from the harm?

The Court concludes that because Plaintiffs have failed to show by a preponderance of the evidence that Dr. Francis failed to meet the standard of care in her handling of Mrs. Gharbi's labor and delivery, judgment will be entered in favor of Defendant. Alternatively, if the Court were to conclude that Dr. Francis breached the standard of care, the Court concludes that Plaintiffs have not shown by a preponderance of the evidence that the breach of the standard of care caused E.G.'s death.

II. Finding of Facts Relevant to Liability

On January 21, 2022, the parties filed a Comprehensive Statement of Agreed Upon Facts Submitted by the Parties ("CSF"). (Doc. 77-1.) The parties also filed post-trial submissions: Plaintiff's Supplemental Findings of Fact and Conclusions of Law (Doc. 116) and Defendant's Proposed Findings of Fact and Conclusions of Law Revised After Trial

(Doc. 117). The Court's findings of fact are derived from these documents as well as the testimony and evidence presented at trial.

A. GLOSSARY OF TERMS

1. **Acidosis** – Acidosis is a rearrangement of the balance of the oxygen, hydrogen, and carbon dioxide in the body. (Hammers, TT Day 2, 20:10-12.) It is a buildup of acids in the blood: "normally, we go through a metabolic pathway that makes energy that does not produce acid because we have sufficient oxygen available but, when there's less oxygen available, our bodies become a different pathway and that other pathway to metabolize and make energy produces acids." (Golen, TT Day 5, 31:15-23.) Acidosis is when hypoxia has persisted for a period of time or is changing the fetus's metabolic status so it is becoming more acidotic which means it becomes metabolically unstable. (Mauldin, TT Day 1, 151:9-13, 218:19-22.) Acidosis can result in neurologic damage or death. (Mauldin, TT Day 1, 155:5-6.) Acidosis is a pH value that is below the normal 7.4 or 7.2 or 7.1. (Mauldin, TT Day 1, 220:21-23.) Lab results showing a pH value of 7.353 for blood collected from a double-clamped sample of cord, within one minute after delivery, would be a normal pH value for a neonate. (Mauldin, TT Day 1, 243:1-6; Pettker, TT Day 4, 52:4-15.)
2. **Arrest of Labor** – Arrest of Labor pursuant to the American College of Obstetricians and Gynecologist ("ACOG") means no change in cervical dilation after four hours despite adequate contractions. (Mauldin, TT Day 1, 169:3-13.) It means that the cervix

does not change at all after six centimeters for four hours with ruptured membranes in the face of adequate contractions. (Golen, TT Day 5, 33:12-14.)

3. **Asphyxia** – Asphyxia is a lack of oxygen to the body. (Hammers, TT Day 2, 20:16-20.)
4. **Bradycardia** – Bradycardia means the heart rate is going below the typical range, which is between 110 and 160, for an extended period and usually refers to something greater than 8 to 10 minutes. With severe bradycardia, the brain does not get the oxygen it needs and then damage starts. (Mauldin, TT Day 1, 155:9-18.) Bradycardia is a fetal heart rate below 110 that has lasted more than ten minutes. (Pettker, TT Day 4, 38:12-14.)
5. **Electronic Fetal Monitoring** – Strips/Tracings
    - Electronic fetal monitoring is a process that follows the fetus's heart rate and the variations in that heart rate over time that allows the provider to assess whether the fetus is tolerating labor well. (Mauldin, TT Day 1, 148:21-149:5.) The fetal monitor is either a monitor on the abdomen of the mother that is able to detect sounds and motion of the fetal heart and graph the fetal heart rate on the monitor or a fetal scalp electrode which detects the electronic impulses of the fetal heart and maps them to the screen as a fetal heart rate. (Pettker, TT Day 4, 13:19-24.) The electronic fetal monitor is used as a window into the physiologic state of the fetus and gives clues as to the oxygenation and then acid-based status of the fetus, information that is used to manage the labor. (Pettker, TT Day 4, 14:2-7.)

- **Accelerations** – Accelerations are visually apparent abrupt increases in the fetal heart rate that generally last 15 seconds or more and are defined as being a 15-beat per minute increase in the fetal heart rate tracings. (Pettker, TT Day 4, 20:19-21:2.) Presence of accelerations is highly indicative of a healthy fetus that is not affected by hypoxia. (Pettker, TT Day 4, 21:5-7.) An acceleration is a momentary rise from the fetal heart rate baseline, and it is a reliable predictor that there's not acidemia, it's the one isolated characteristic of the fetal heart rate tracing that gives us reliable information that there's not acidemia. (Golen, TT Day 5, 38:17-21.)
- **Decelerations** - Decelerations are visually apparent decreases in the fetal heart rate tracing, and there are three types of decelerations: early decelerations, late decelerations, and variable decelerations.[1] (Pettker, TT Day 4, 18:5-8.) An early deceleration mirrors the pattern of the contraction, starting at the beginning of the contraction and ending at the end of the contraction and the physiology behind it is believed to be head compression—the heart rate slows when the head is compressed. (Pettker, TT Day 4, 19:4-9.) A variable deceleration is an abrupt increase and abrupt decrease in the fetal heart rate that sometimes happens with

[1] Dr. Pettker noted that the Practice Bulletin of the American College of Obstetricians and Gynecologists ("ACOG") "has very objective definitions of what decelerations are in terms of being visually apparent and for early, late, and variables, the time of onset, the appearance of them, whether or not it's a gradual decrease or an abrupt decrease, all of those are described really clearly in the ACOG Practice Bulletin." (TT Day 4, 18:12-17.)

contractions and sometimes between contractions and the physiology behind it is believed to be cord compression. (Pettker, TT Day 4, 19:11-16.) A late deceleration is believed to be because of uteroplacental insufficiencies or decrease in the placenta or the mother to deliver oxygen to the fetus. (Pettker, TT Day 4, 19:18-21.) "Subtle deceleration" and "repetitive deceleration" are not accepted terms and are not described in the literature. (Pettker, TT Day 4, 19:22-20:18.) Deceleration is some change from the baseline of wherever the heart rate was staying, in most instances between 110 and 160 but with deceleration it's going to fall by 15 beats at least, typically by 20 or 30 beats and stays that way for 30 seconds to a minute or more. (Mauldin, TT Day 1, 154:1-6.) When the deceleration comes with the contraction it is called a variable deceleration which the fetus can tolerate a bit longer than a late deceleration--when the deceleration comes after the contraction it reflects decreased oxygen to the baby. (Mauldin, TT Day 1, 154:7-11.)

> When contractions start, that's pressure on the uterus, that's squeezing the baby and perhaps squeezing the umbilical cord, and that pressure then potentially can decrease the blood flow through the umbilical cord to the baby, so in those instances, it might cause fetal heart rate decelerations. So that's particularly what we look for, because if there's repetitive decelerations or decelerations that start to come after the contractions, it can show us that the baby is becoming hypoxic or not getting as much oxygen as it can or as it should through the placenta, and hypoxia, if it goes on for a long period of time, could result in acidosis.

(Mauldin, TT Day 1, 149:14-25.)

- **Variability** – Variability refers to the fluctuations in the baseline fetal heart rate that can be described in four categories: 1) absent is a totally straight line, totally undetectable variability;[2] 2) minimal would be a variation that is between one and five beats per minute; 3) moderate is between six and twenty-five beats per minute; and 4) marked is more than twenty-five beats per minute. (Pettker, TT Day 4, 16:24-17:10.) Variability is a reflection of the neurologic and cardiovascular state of the fetus and can be impacted by the acid-based status, whether the fetus is asleep, medications, and baseline neurologic status. (Pettker, TT Day 4, 17:14-22.) "Variability is seeing the heart rate go up and down . . . vary[ing] between 5 and 25 beats over 5 to 15 seconds, so we see just a lot of variation in the heart rate. . . . [H]ealthy babies are going to have moderate variability, that means their heart rate varies a good bit like between 6 and 25 beats per minute." (Mauldin, TT Day 1, 154:17-20.) "[A]s it becomes minimal variability, which means it's between 0 and 5 beats of variation . . . we need to be watching more closely." (Mauldin, TT Day 1, 154:20-23.) Loss of variation indicates "that the baby is struggling, in some way, perhaps, becoming hypoxic, which we would be concerned about, ultimately, leading to the acidosis." (Mauldin, TT Day 1, 150:5-12.)

[2] Dr. Golen said that absent variability "is the straightest line that you can possibly imagine." (TT Day 5, 28:25-26:2.)

- **Fetal Strip Categories** – There is a three-tiered interpretive system that is used for interpreting fetal heart rate monitoring.[3] (Pettker, TT Day 4, 14:23-25.) A branch of the National Institutes of Health ("NIH") defines the categories and the definition is endorsed by the American College of Obstetrics and Gynecology. (Golen, TT Day 5, 27:16-24.) Fetal strips are also called fetal tracings. (*See, e.g.*, Pettker, TT Day 4, 15:8-9.)
    - Category I Strip – A Category I strip is a very basic strip that indicates the fetus is well-oxygenated, very healthy, good variations in the heart rate and, if there is a deceleration, it is what we consider to be an early or a variable type and it is happening infrequently. (Mauldin, TT Day, 1 155:20-35.) This category suggests a healthy or normal heart-rate pattern and that would be a baseline of the heart rate between 110 and 160 beats per minute, that there's moderate variability present, there's the absence of late or variable decelerations, and there may be accelerations. (Pettker, TT Day 4, 15:1-6.)
    - Category II Strip – A Category II strip is everything that is not Category I or Category III; it is an intermediate category where you need to make decision points frequently including whether the fetus is healthy enough to

[3] Dr. Pettker noted that his definitions are generally from the ACOG, the American College of Obstetricians and Gynecologists, and the NICHD, National Institute for Child Health and Development. (TT Day 4, 16:5-7.)

continue laboring for another thirty minutes or an hour and will the baby be delivering soon. (Mauldin, TT Day 1, 156:4-11.) A Category II strip has a wide range of interpretation and meaning, and Category II tracings are the most common tracings in normal births. (Pettker, TT Day 4, 15:24-25.)

- Category III Strip – A Category III strip is when the tracing has lost variability, meaning absent variation, and it can be that alone or also have repetitive variable and late decelerations. (Mauldin, TT Day 1, 156:1-4.) A Category III strip is suggestive of a fetus that may be at risk and is characterized as absent baseline variability, an absolutely flat line, because it is entirely undetectable variability to the tracing. (Pettker, TT Day 4, 15:8-12.) It would also have to have recurrent late decelerations, recurrent variable decelerations, or a bradycardia. (Pettker, TT Day 4, 13-15.) A Category III strip has absent baseline in the presence of any of those three conditions—recurrent late decelerations, recurrent variable decelerations or a bradycardia with a deceleration that occurs with 50 percent or more of the contractions during that period of time. (Pettker, TT Day 4, 16-21.)
- It is very common in clinical practice and well-established in the literature that there is inter-observer variability in interpretation of fetal heart rate monitoring. (Pettker, TT Day 4, 38:2-5.) It can make a difference whether

the fetal heart rate tracings are being interpreted in real-time or after the fact because the determination or conclusion about what a fetal heart rate tracing represents is influenced by the outcome when it's known so that retrospective review of fetal heart rate tracings is biased by the outcome. (Golen, TT Day 5, 30:24-31:7; Pettker, TT Day 5:5-8.)

6. Hypoxia –Hypoxia is decreased oxygenation to the body. It can lead to asphyxia if it is prolonged over a period of time and can reach a critically low level that basically stops the blood supply. (Hammers, TT Day 2, 20:22-21:2.) "Hypoxia [is] a loss of the oxygen concentration in the blood, and . . . if the baby persists in having a low oxygen concentration, . . . it progresses into acidemia or acidosis, which means it becomes metabolically unstable, so it becomes more acidotic. . . . Over time, as the degree of acidosis worsens, the outcome for the baby becomes much more guarded or at risk for having long-term problems." (Mauldin, TT Day 1, 151:9-16.) Fetal heart rate monitoring is the only way a provider can assess whether there's been some intrapartum asphyxia, hypoxia, or acidosis. (Mauldin, TT Day 1, 151:21-24.) Long-term hypoxia begins to make the fetus acidotic and it's the acidosis that is the risk for causing long-term harm. (Mauldin, TT Day 1, 218:15-18.) Put differently, acidosis is when hypoxia has persisted for a period of time or is changing the fetus's metabolic status so it's becoming more acidotic. (Mauldin, TT Day 1, 218:19-21.) Acidosis is a pH value that is below the normal, 7.4, or 7.2, or 7.1. (Mauldin, TT Day 1, 220:21-23.) The objective measure of

whether there is acidosis is at the time of delivery is the cord gas indicating what the pH value of the baby's blood is. (Mauldin, TT Day 1, 220:17-20.)

7. Polyhydramnios – In this case polyhydramnios means much amniotic fluid. (Chung, TT Day 3, 69:18-19.) Reasons for it can be that the fetus is not swallowing: amniotic fluid is actually fluid that is recycled, it comes from the kidneys, and essentially, is urinated out and gets swallowed and gets recycled through the body, so that builds up if the fetus is not swallowing normally. (Chung, TT Day 3, 69:19-25.)
8. Tachycardia – Tachycardia is when the adult's heart rate goes over 100 beats per minute. (Francis, TT Day 1, 102:5-8.)
9. Tachysystole – Tachysystole is more than five contractions in a ten-minute period which, in many instances, is considered too much uterine activity. (Mauldin, TT Day 1, 185:17-23.)

B. SUMMARY OF SEQUENCE OF EVENTS

10. Plaintiffs, Imen Gharbi and Hattab (Ben) Gharbi are the parents of E.G. who died on December 18, 2017. (CSF ¶ 1.)
11. At that time, Potacia W. Francis, M.D., was Mrs. Gharbi's attending obstetrician at Harrisburg Pinnacle Hospital where Mrs. Gharbi was induced. (CSF ¶ 2.)
12. Medical records indicate that Mrs. Gharbi's due date was December 9, 2017. (CSF ¶ 3.)

13. Gerald Maenner, M.D., a board-certified obstetrician and gynecologist was Mrs. Gharbi's prenatal obstetrician. (PFF ¶ 5.)

14. On or about December 16, 2017, prenatal records document that Mrs. Gharbi was overdue to deliver and was 41 weeks pregnant. (CSF ¶ 5.)

15. On December 16, 2017, Mrs. Gharbi was admitted to the hospital to induce labor. (CSF ¶ 6.)

16. Dr. Maenner was the attending physician, and he was assisted by OB-GYN resident Megan Klamerus, D.O. (PFF ¶ 12.)

17. Hospital records document that, upon admission, Mrs. Gharbi' s cervical exam was 2 centimeters and the fetal head was ballotable which means that the head was still fixed in the pelvis and was not yet engaged in the birth canal. (CSF ¶ 7; PFF ¶¶ 12-13.)

18. Induction of labor was started at 3:00 p.m. on December 16, 2017, with a single dose of Cytotec (50 mcg). (CSF ¶ 8; PFF ¶ 14.)

19. Hospital records document that five hours later, at approximately 8:00 p.m., Mrs. Gharbi had a 1 centimeter change in dilation from 2 centimeters to 3 centimeters. The fetal head was still ballotable, Mrs. Gharbi had contractions every 2 to 4 minutes, and fetal monitoring documented a Category II strip. (CSF ¶ 9.)

20. Hospital records document that induction efforts to induce labor continued with IV Pitocin (1 milli-unit) starting at approximately 11:29 p.m. on December 16, 2017. (CSF ¶ 10.)

21. Medication administration records document that at 1:00 a.m. on December 17, 2017, Pitocin administration was stopped. (CSF ¶ 11.) Hospital records document that Mrs. Gharbi was feeling strong contractions and Pitocin was stopped because of late decelerations. (CSF ¶ 12.)

22. A report entered by Dr. Klamerus indicates that she documented a Category II strip with notations that, as of 7:22 that morning, the fetal tracings still had late decelerations, there was no variability in the fetal strip, there were no accelerations, and the plan was to restart Pitocin. (TT Day 1, 37:1-19, 41:23-25.)

23. Shift change and sign out to Dr. Francis took place at approximately 8:00 a.m. on December 17, 2017. (CSF ¶ 17.) At that time, Dr. Francis was employed by a federally funded clinic and was covering a weekend shift at the hospital. (CSF ¶ 14.)

24. According to the medication administration record, Pitocin remained off at the time of the 8:00 a.m. sign out to Dr. Francis. (CSF ¶ 16.)

25. Dr. Maenner, Dr. Klamerus, Dr. Francis, and Dr. Franck Lin, an OB-GYN resident, were present at the sign out. (CSF ¶ 18.)

26. At shift change and sign out, Dr. Francis, as the attending physician, assumed responsibility for the care of Mrs. Gharbi and her unborn baby. (CSF ¶ 20.)

27. According to Dr. Francis, sign out is very comprehensive and entails the providers, "relating the course of the previous care - the care that happened on

the previous shift, anything that's pending or needs to be addressed, anything . . . that's in the process of being evaluated and needs to be completed." (CSF ¶ 19.)

28. Dr. Klamerus completed her 7:22 a.m. note at 8:41 a.m. and indicated that "we will restart low dose Pitocin." (TT Day 1, 86:17-24.)

29. At 12:46 p.m. on December 17, 2017, Dr. Lin documented Mrs. Gharbi as 6 centimeters dilated, the fetal head as still ballotable, and fetal monitoring revealed a Category II strip due to minimal fetal heart rate variability. (CSF ¶ 21.)

30. Medical records document Dr. Lin planning to re-start Pitocin. (CSF ¶ 22.)

31. Records also document that Dr. Francis signed off on Resident Lin's note at 2:23 p.m. on December 17, 2017. (CSF ¶ 23.)

32. The medication administration record documents that Pitocin was restarted at 2:31 p.m. (1 milli-unit) and increased to 2 milli-units at approximately 3:00 p.m. (CSF ¶ 24.)

33. Hospital records document that at 3:00 p.m. on December 17th Dr. Francis was the "OB examiner," Mrs. Gharbi was 7 centimeters dilated, and Dr. Francis artificially ruptured Mrs. Gharbi's membranes. (CSF ¶ 25; PFF ¶ 93.)

34. Thereafter, at 3:30 p.m., the medication administration record indicates that Pitocin was increased to 4 milli-units and again increased to 6 milli-units by 4:00 p.m. (CSF ¶ 26.)

35. At approximately 7:00 p.m. medical records document that Mrs. Gharbi had remained dilated at 7 centimeters despite resumed Pitocin induction at 4 to 6 milli-units and contractions every 4 to 6 minutes. (CSF ¶ 27.)

36. Hospital records document that at approximately 7:30 p.m. Mrs. Gharbi was tachycardic (117 bpm), and her blood pressure was flagged as abnormal (122/95). (CSF ¶ 28.)

37. Hospital records document that nurses initiated various fetal resuscitative measures from 7:40 p.m. to 7:44 p.m., including turning Pitocin off at 7:41 p.m., repositioning Mrs. Gharbi, administering oxygen to her, and increasing IV fluids. (CSF ¶ 29.)

38. Dr. Francis was notified and arrived at her bedside at 7:45 p.m. (CSF ¶ 30; PFF ¶ 124.)

39. Resident Samantha Nguyen, M.D., documented a fetal heart rate in the 50's. (CSF ¶ 31.)

40. Hospital records document that labor and delivery nurses noted a "14- minute deceleration" at approximately 8:00 p.m. on December 17th. (CSF ¶ 32.)

41. Dr. Francis thought that the "[nursing] interventions had worked, and the tracing had recovered." (CSF ¶ 33.)

42. She left Mrs. Gharbi's bedside and attended to other patients. (CSF ¶ 34.)

43. Between 8:00 p.m. and 9:00 p.m., medical records documented Mrs. Gharbi as tachycardic. (CSF ¶ 35.)

44. Medical records document that at 9:00 p.m. fetal heart tracings revealed a baseline of 140 bpm with "minimal: undetectable to 5 bpm" variability. (CSF ¶ 36.)

45. At 9:16 p.m., nurses initiated fetal resuscitative measures, including repositioning Mrs. Gharbi and administering supplemental oxygen via non-rebreather. (CSF ¶ 37.)

46. At 9:30 p.m., fetal heart tracings revealed a baseline of 140 bpm with variable decelerations, late decelerations, and "minimal: undetectable to 5 bpm" variability. (CSF ¶ 38.)

47. Hospital records document that Mrs. Gharbi remained tachycardic at 9:41 p.m. (139 bpm at that time and 126 bpm at 9:55 p.m.). (CSF ¶ 39.)

48. Hospital records document that fetal heart rate tracings revealed baseline 125 bpm with "minimal: undetectable to 5 bpm" variability by 10:00 p.m. (CSF ¶ 40.)

49. Hospital records document that Mrs. Gharbi's pulse rate remained tachycardic at 10:11 p.m. (CSF ¶ 41.)

50. Hospital records document that Dr. Nguyen was notified of Mrs. Gharbi's tachysystole at 10:18 p.m. (CSF ¶ 42.)

51. Hospital records document that Mrs. Gharbi was still tachycardic (148 bpm), and that fetal heart tracings revealed a baseline of 125 bpm with "minimal: undetectable to 5 bpm" variability at approximately 10:30 p.m. (CSF ¶ 43.)

52. Hospital records document that Dr. Francis and Dr. Nguyen were at Mrs. Gharbi's bedside at 10:51 p.m. (CSF ¶ 44.)

53. Dr. Francis did not initiate delivery because she believed Mrs. Gharbi was close to delivering at that time. (CSF ¶ 45.)

54. Medical records document that Mrs. Gharbi started pushing at 10:54 p.m. (CSF ¶ 46.)

55. Dr. Francis left Mrs. Gharbi's bedside while she pushed. (CSF ¶ 47.)

56. Medical records indicate that Mrs. Gharbi was administered supplemental oxygen via non-rebreathermask at 11:28 p.m. (CSF ¶ 48.)

57. Medical records document that fetal tracings revealed a baseline of 115 bpm, variable decelerations and "minimal: undetectable to 5 bpm" variability at 11:30 p.m. (CSF ¶ 49.)

58. By midnight on December 17, 2017, medical records indicate that fetal heart rate tracings revealed baseline 120 bpm, variable decelerations and "minimal: undetectable to 5 bpm" variability. (CSF ¶ 50.)

59. Between midnight and 1:00 a.m. on December 18, 2017, medical records document that Mrs. Gharbi remained tachycardic. (CSF ¶ 51.)

60. At 1:00 a.m. fetal heart rate tracings demonstrated baseline 120 bpm, early and variable decelerations and "minimal: undetectable to 5 bpm" variability. (CSF ¶ 52.)

61. Hospital records document that at approximately 1:00 a.m. on December 18, 2017, after two (2) hours of pushing with no descent and the baby's head still not engaged, Dr. Francis ordered a cesarean section. (CSF ¶ 53.)

62. Medical records document that the Gharbis' baby was delivered at 1:41 a.m. on

December 18, 2017. (CSF ¶ 54.)

63. Medical records indicate that a NICU physician, Ecaterina Sartina, M.D., was called to Mrs. Gharbi's delivery room and she documented that it was because of "fetal distress with symptoms of variable decelerations." (TT Day 1, 71:8-12.)

64. Medical records document that the NICU physician arrived within 1 to 2 minutes of the E.G.'s birth during active CPR in progress. (CSF ¶ 56.)

65. Medical records reflect that E.G.'s initial APGAR score was zero, he was unresponsive, had no cry, had no heartbeat, no pulse, was not breathing, he displayed poor profusion, and he was pale. (CSF ¶ 58.)

66. Medical records document that, with resuscitation efforts, there was a heartbeat of 30-40 bpm during the chest compressions but no pulse on the femoral arteries. (CSF ¶ 60.)

67. Medical records document that E.G.'s APGAR scores were zero at 1 minute, 1 at 5 minutes, 1 at 10 minutes, zero at 15 minutes, zero at 20 minutes, and resuscitative efforts were terminated at 2:00 a.m. on December 18, 2017. (CSF ¶ 61.)

68. The umbilical cord blood collected was a venous sample. (CSF ¶ 63.)

69. E.G.'s autopsy was carried out by the hospital's pathologist. (CSF ¶ 65.)

70. In the clinical summary of the autopsy report, the pathologist reported that E.G. showed signs of stress and that an emergency C-section was carried out. (CSF ¶ 66.)

71. E.G.'s genetic testing revealed a normal male karyotype without structural

abnormality or rearrangement. (CSF ¶ 67.)

72. There were 23 pairs of chromosomes and each of E.G.'s chromosomes appeared normal. (CSF ¶ 68.)

73. The medical records document that both Mr. and Mrs. Gharbi subsequently underwent cytogenetic testing themselves and both Imen and Hattab (Ben) Gharbi's karyotype results were normal. (CSF ¶ 69.)

74. At autopsy, the pathologist documented E.G.'s skin appearance as unremarkable, there was no skin slipping and no necrosis, there was no subcutaneous edema, head and neck exam, chest and abdominal contours and external genitalia were all unremarkable, and the upper and lower extremities demonstrated contractures. (CSF ¶ 70.)

75. Autopsy findings further demonstrated no organomegaly, no significant peritoneal fluid, abdominal and pelvic organs were normal in locations and gross appearances, and the lungs were normal in appearance, slightly firm and did not float in water. (CSF ¶ 71.)

76. The autopsy listed possible Hirschsprung's Disease and noted limb contractures. (CSF ¶ 72.)

77. The autopsy pathologist commented that several congenital syndromes can be associated with both Hirschsprung's Disease and limb contractures but skeletal abnormalities are generally much more pronounced than what he saw in E.G.'s case.

(CSF ¶ 73.)

78. The hospital's record documented Baby Gharbi's final diagnosis as an ICD/10/P84 code, which pertains to newborn problems relating to acidemia, acidosis, anoxia, asphyxia, hypercapnia, hypoxemia, hypoxia, and mixed metabolic and respiratory acidosis. (CSF ¶ 74.)

79. The medical examiner at Dauphin County Coroner's Office did not perform the autopsy. (CSF ¶ 75.)

80. The death certificate signed by the chief deputy coroner listed complications of Hirschsprung Disease and "Probable Genetic Disorder" as the causes of death. (CSF ¶ 76.)

C. FACT WITNESS TESTIMONY

- **TESTIMONY OF POTATIA FRANCIS, M.D.**

81. Dr. Francis learned about Mrs. Gharbi at sign out. (TT Day 1, 20:1-5.)

82. Dr. Francis said she received information orally at sign out from Dr. Maenner and Dr. Klamerus that Mrs. Gharbi was admitted for an induction, that she was being induced, that she was on Pitocin, that she had not been told Pitocin had been stopped. (*See, e.g.*, TT Day 1, 26:1-3, 25.)

83. Dr. Francis agreed that Pitocin is used to stimulate contractions, that it can stimulate stronger and faster contractions, that stronger and faster contractions can reduce blood and oxygen flow to cause the baby to experience abnormal fetal heart rate patterns if

certain parameters are exceeded, the fact that Pitocin had been stopped was significant, medication information was available to her in the chart, and stopping Pitocin is one of the first interventions used to resuscitate a fetus that is in distress. (TT Day 1, 29:15-25, 31:22-25, 32:1-4, 33:12-16.)

84. Had Dr. Francis known that Pitocin had been stopped because of late decelerations, she would not have performed a caesarian section the morning of sign out but would have talked with Mrs. Gharbi about delivery options. (TT Day 1, 35:25-36:3.)

85. Just before sign out, Dr. Francis said that Dr. Klamerus had documented a Category II strip with notations that, as of 7:22 that morning, the fetal tracings still had late decelerations, there was no variability in the fetal strip, there were no accelerations, and the plan was to restart Pitocin. (TT Day 1, 37:1-19, 41:23-25.)

86. Dr. Klamerus completed her 7:22 a.m. note at 8:41 a.m. and indicated that "we will restart low dose Pitocin." (TT Day 1, 86:17-24.)

87. Based on the plan recorded by Dr. Klamerus, Dr. Francis stated that the plan of care at the time of sign out indicated that Mrs. Gharbi was on Pitocin. (TT Day 1, 41:25-42:2, 87:3-5.)

88. Dr. Francis agreed that a patient with a Category II strip requires close monitoring. (TT Day 1, 38:1-6.)

89. Dr. Francis said that she relied on residents Dr. Lin and Dr. Nguyen to tell her what was happening with Mrs. Gharbi and they then collaborated on the management of her care.

(TT Day 1, 39:24-40:7.)

90. She also said that she relied on the labor and delivery nurses to tell her what was going on with Mrs. Gharbi's labor course. (TT Day 1, 40:8-10.)

91. The residents rather than the attendants write the plan of care in the chart. (TT Day 1, 41:3-17.)

92. Dr Francis indicated that she met the Gharbis in the morning on December 17th and, when she initially saw Mrs. Gharbi, she thought her strip was a Category I. (TT Day 1, 98:7-12.)

93. Looking at the tracings from about 9 and 9:30 in the morning, Dr. Francis confirmed that she interpreted the strips as Category I and also stated that Mrs. Gharbi had Category I fetal heart rate tracings throughout the course of the day from time to time. (TT Day 1, 98:13-24.)

94. Dr. Francis agreed that Dr. Maenner or the residents never documented a Category I strip, and Dr. Klamerus noted that the fetus had a category II strip at all times, but she said that Petronella Voorhoog, R.N., the nurse who restarted Pitocin at 2:31 p.m. on the 17th, had documented that the patient was having an active fetus with movements and moderate variability with no decelerations which is, in essence, a Category I strip. (TT Day 1, 119:24-121:8.)

95. After sign out, the first recorded physician encounter with Mrs. Gharbi was at 12:46 p.m. when Dr. Lin reported that, after several checks, the baby continued to have a ballotable

fetal head, the strip was Category II because of minimal fetal heart rate variability, and he was planning to restart Pitocin. (TT Day 1, 43:22-44:18.)

96. Pitocin was restarted at 2:31 p.m. (TT Day 1, 50:18-51:5.)

97. By 3:00 p.m., Pitocin was increased to 2 milliunits; by 3:30, it was increased to 4 milliunits; and, by 4:00, it was increased to 6 milliunits. (TT Day 1, 52:5-11.)

98. Although Dr. Francis stated that she had seen Mrs. Gharbi in the morning when she came on shift, the first documented encounter was at 3:00 p.m. (TT Day 1, 52:13-19.)

99. Dr. Francis also stated that it is the nurse in the room who documents the encounter so if she goes into a room when the nurse is not there the encounter will not be documented. (TT Day 1, 53:7-14.)

100. Based on information provided by Dr. Francis, at 3:00 p.m. the nurse in the room recorded that Mrs. Gharbi was 7 centimeters dilated and Dr. Francis ruptured Mrs. Gharbi's membranes. (TT Day 1, 53:22-54:9.)

101. Dr. Francis agreed that the 7:00 p.m. note recorded by Dr. Nguyen indicates that Mrs. Gharbi was still 7 centimeters dilated and a nurse recorded that Mrs. Gharbi was 7 centimeters dilated at 6:42 p.m., noting that there had been no change in cervical dilation. (TT Day 1, 53:10-54:20.)

102. Dr. Francis testified that, during the 7 to 8 p.m. period, Mrs. Gharbi had Category I and Category II fetal heart tracings before there was a period of marked variability and then loss of contact. (TT Day 1, 103:3-8.)

103. By "loss of contact" she meant "the baby is off the monitor, for some reason, the nurses are not able to document the heartbeat." (TT Day 1, 103:5-15.)

104. Dr. Francis also agreed that at 7:38 p.m., with Dr. Nguyen present, Mrs. Gharbi was being turned to different positions, at 7:39 she was being given oxygen by a non-rebreather mask, at 7:40 she was being turned on her sides, knees put up to her chest, I.V. fluids were increased, and additional nurses were at her bedside, and at 7:41, Pitocin was turned off. (TT Day 1, 56:16-57:11.)

105. Dr. Francis stated that these actions were taken because "they could not document the fetus, the fetus was off the monitor." (TT Day 1, 57:12-15.)

106. Dr. Francis arrived in Mrs. Gharbi's room at 7:45. (TT Day 1, 57:16-18.)

107. When she arrived in the room, others were attempting to put Mrs. Gharbi back on the monitor and resuscitate the fetus. (TT Day 1, 103:19-22.)

108. It was Dr. Francis's understanding "that the monitors were malfunctioning, and the Resident tried to do a scalp monitor, and that wasn't working as well, and then the second scalp monitor was in place and that started to work." (TT Day 1, 103:25-104:4.)

109. Dr. Francis explained that a scalp monitor attaches the scalp electrode to the fetus's head through the cervical opening attached directly to the fetus instead of placing the monitor that transmits sound waves on the patient's abdomen. (TT Day 1, 104:6-13.)

110. Dr. Francis testified that once the fetal scalp monitor was functioning, the fetal heart rate was documented, the fetal heart rate came back to baseline with moderate

variability, and the fetus was resuscitated, she felt that the interventions the nurses had conducted were adequate, there was no need for further intervention, and there was no indication that she needed to initiate a plan to deliver the baby at that time. (TT Day 1, 58:19-59:7, 104:15-105:14.)

111. Thereafter, Dr. Francis said left the room and oxygen continued to be administered at 8:00 because oxygen is not stopped right away but rather left in place for a while. (TT Day 1, 59:9-60:23.)

112. Dr. Francis agreed that at 8:00, 8:30, and 9:00, the nurse wrote that the fetus had minimal to undetectable heart rate variability, and at 9:30 p.m. the nurse wrote that the fetus also had variable decelerations and late decelerations, but Dr. Francis said that her assessment was different in that she found the tracings (which can be seen on monitors outside the patient's room) showed periods of moderate variability and periods of accelerations (an indicator of fetal well-being and good fetal acid base status or pH status). (TT Day 1, 61:13-64:7.)

113. The nurse continued to record minimal to no heart rate variability through 10:00 p.m., and Dr. Nguyen documented tachysystole at 10:18. (TT Day 1, 64:20-65:5.)

114. Dr. Francis said she did not initiate a plan to deliver the baby at that time because she believed that Mrs. Gharbi was going to deliver shortly. (TT Day 1, 66:1-7.)

115. At 11:00 p.m., the nurse again recorded minimal to undetectable fetal heart rate, an assessment with which Dr. Francis disagreed. (TT Day 1, 66:12-17.)

116. At that time, early and late decelerations were also noted. (TT Day 1, 67:13-15.)

117. Dr. Francis explained that this was at a time when Mrs. Gharbi was pushing, the early and late decelerations were not abnormal with pushing and not worrisome as long as it's not recurrent, adding that the fetal heart tracing in the record did not show minimal variability and showed some accelerations occurred. (TT Day 1, 67:16-69:4.)

118. At 1:00 a.m. on December 18, 2017, Dr. Francis discussed having a cesarean section and Mrs. Gharbi agreed to it. (TT Day 1, 71:1-2.)

119. Dr. Francis recommended a cesarean because Mrs. Gharbi had been pushing for a while, and when Dr. Francis saw her at 1:00 she was very tired and the head was not yet engaged so she still had lot more pushing to do and she was too tired to proceed. (TT Day 1, 107:17-108:19.)

120. Fetal heart monitors are disconnected when the patient leaves the delivery room--once the patient gets to the operating room and before the patient is prepped for surgery, the nurses listen with a Doppler handheld machine to check the status of the fetus. (TT Day 1, 109:23-110:17.)

121. The Doppler measurement in this case was taken at 1:28 a.m. and the record indicated 120 bpm which Dr. Francis described as a normal rate for a fetus in that it is within the range of 110 to 160 beats per minute. (TT Day 1, 111:2-15.)

122. She stated that if the Doppler reading documented that there was no heartbeat,

you would do an emergency C-section and, if it's normal, you proceed as planned with a non-emergency C-section. (TT Day 1, 111:23-112:3.)

123. E.G. was delivered at 1:41 a.m. (TT Day 1, 71:5-7.)

124. Medical documentation indicates that cord venous blood was collected on December 18th at 2:41 a.m.[4] (TT Day 1 71:19-23.)

125. Dr. Francis stated "since I was getting cord gases, [the circulator] would hand us two additional clamps so we will clamp further down . . . and cut in between and give a segment of the cord off to the circulator, who then hands it to the nurse in the room for her to collect cord gases," a process which happens in "rapid succession, it's within a minute." (TT Day 1, 113:13-24.) She also said that, after she hands off the cord, she has no further involvement as to where or how the sample is processed. (TT Day 1, 72:3-13.)

126. Dr. Francis explained that the documentation that the cord was clamped and cut means that the cord was double clamped because, "if its single, it will exsanguinate the baby." (TT Day 1, 72:21-73:1.)

127. The following exchange took place concerning E.G.'s appearance immediately upon delivery, beginning with Dr. Francis's description of what he looked like at that time: A. Well, when we took him out, he was very stiff. Usually, the babies are limp, but

[4] The meaning of "collected on" and other terms found on relevant medical test result documentation are addressed later in the text by fact witness Brooke Crummel.

he was very stiff, his limbs are what we call contracted, so his arms were bent and, kind of, folded in, his legs were also bent and folded in, you couldn't move them, they were very stiff, and his chest was kind of sunken and, kind of, just sunken and abnormal in shape.

> Q. Could you tell if he was breathing?
>
> A. He gasped, he gasped, initially. We clamped the cord and we handed him off.
>
> Q. What reaction did you have to the baby's condition?
>
> A. I was startled, I was startled. I was just shocked at his appearance, and that's why I got the cord gases. I was just shocked. I've practiced for a long time and I've never delivered a baby that appeared that way.

(TT Day 1, 115:6-19.)

128. Dr. Francis was not involved in the resuscitation efforts of E.G. as she was still operating on Mrs. Gharbi. (TT Day 1, 74:14-16.)

129. Although she authorized genetic testing for the Gharbis, Dr. Francis was not involved in their care after her shift ended on December 18, 2017. (TT Day 1, 77:10-12, 77:25-78:1.)

- **TESTIMONY OF GERALD MAENNER, M.D.**

130. Gerald Maenner, M.D., whose deposition testimony was read into the record, is board-certified in obstetrics and gynecology and, at the relevant time, saw patients at the UPMC Pinnacle Health Women's Outpatient Center and Harrisburg Hospital. (Maenner

Dep. 6:16-7:2, Pls.' Ex. 9.)

131. He had provided prenatal care to Mrs. Gharbi. (Maenner Dep. 10::5-8.)

132. Dr. Maenner verified that Mrs. Gharbi had an ultrasound at about thirty-two weeks (October 2017) indicating she had an amniotic fluid index of 24.1 which was upper normal amniotic fluid. (Maenner Dep. 9:24-10:17.)

133. There was no diagnosis of polyhydramnios at that time. (Maenner Dep. 10:18-22.)

134. Another ultrasound at approximately thirty-six weeks indicated an amniotic fluid index of 33.7 which the report indicated was moderately increased polyhydramnios. (Maenner Dep. 11:24-12:3.)

135. Dr. Maenner did not recall any treatment for the 33.7 level of fluid. (Maenner Dep. 6-9.)

136. At approximately thirty-nine weeks, Mrs. Gharbi's amniotic fluid index was 14.10 which is normal. (Maenner Dep. 12:17-22.)

137. A week later, the amniotic fluid index was again normal with a reading of 11.95. (Maenner Dep. 13:4-9.)

138. On December 15, 2017, the day before Mrs. Gharbi was scheduled to be induced, the amniotic fluid index was 17.36, a normal value. (Maenner Dep. 13:18-14:3.)

139. Dr. Maenner testified that the one measurement of polyhydramnios that Mrs. Gharbi had at approximately thirty-six weeks had resolved over the course of her last

trimester. (Maenner Dep. 6-14.)

140. Upon admission to the hospital for induction on December 16, 2017, Dr. Maenner and resident Megan Klamerus, D.O., were participating in Mrs. Gharbi's care. (Maenner Dep. 14:20-15:14.)

141. On December 16th and December 17th, Dr. Maenner was supervising Dr. Klamerus. (Maenner Dep. 7:3-15.)

142. At 11:32 p.m., Dr. Klamerus documented that Mrs. Gharbi had no cervical change after one dose of Cytotec, the electronic fetal monitoring strip was a Category II with minimal variability, occasional late decelerations, some accelerations, and the plan was to try introducing Pitocin. (Maenner Dep. 15:1-22.)

143. Dr. Maenner agreed with Dr. Klamerus's assessment of the fetal monitoring strip. (Maenner Dep. 15:11-22.)

144. The December 17, 2017, 1:57 a.m. progress note indicates that there was a reaction of strong contractions on 1 milli-unit of Pitocin, Pitocin was stopped because of late decelerations, and the strip was a Category II strip along with late decelerations. (Maenner Dep. 16:4-17:12.)

145. Dr. Maenner verified that at 1:57 a.m., Mrs. Gharbi was four centimeters dilated and ninety percent effaced, the fetal head was still ballotable, and neither he nor Dr. Klamerus restarted Pitocin thereafter. (Maenner Dep. 18:1-10.)

146. The note recorded at 7:22 a.m. indicates that Mrs. Gharbi was feeling

contractions, there were fetal decelerations, she was five centimeters dilated and fully effaced, the fetal head was still ballotable, and she still had a Category II strip. (Maenner Dep. 19:5-20:1.)

147. Regarding the 8:00 a.m. sign out to Dr. Francis, Dr. Maenner testified that he had no specific recollection of the information passed along to Dr. Francis but, in a typical sign out, the fact that Pitocin had been stopped because of late decelerations would have been discussed to let the incoming team know that there were some concerns about the contraction pattern and Dr. Maenner and Dr. Klamerus did not feel a need to restart Pitocin because the cervix was changing on its own without it. (Maenner Dep. 21:1-22:6.)

148. Dr. Maenner stated that a cesarean section was not warranted at any time that he was caring for Mrs. Gharbi, and he was not involved in any aspect of Mrs. Gharbi's care after the sign out to Dr. Francis on the morning of December 17th. (Maenner Dep. 28:19-29:22.)

- **TESTIMONY OF MEGAN KLAMERUS, D.O.**

149. Megan Klamerus, D.O., whose deposition testimony was read into the record, testified that she was a second year OB-GYN resident doing a rotation at Harrisburg Pinnacle Hospital on December 16, 2017, working under the supervision of Dr. Gerald Maenner when Mrs. Gharbi was admitted for induction. (Klamerus Dep. 6:20-9:9, Pls.' Ex. 8.)

150. Dr. Klamerus explained that whenever a provider opens a note in the

computer, the computer time stamps the note and once the provider signs the note, the computer time stamps it again. (Klamerus Dep. 11:10-12.)

151. Usually, the patient is assessed and then the note is written indicating the results of the physical exam and the plan based on the assessment. (Klamerus Dep. 11:18-22.)

152. Dr. Klamerus confirmed that, following the entry of the December 16th 11:32 p.m. note, Pitocin was started at 11:39 p.m. with the agreement of Dr. Maenner. (Klamerus Dep. 15:3-17:12.)

153. Pitocin was stopped at 1:00 a.m. on December 17th because Mrs. Gharbi was having some late decelerations. (Klamerus Dep. 17:16-18, 18:15-16.)

154. Dr. Klamerus's note opened at 1:57 a.m. and closed out at 3:11 a.m. indicates that Mrs. Gharbi's contraction pattern was irregular, she was four centimeters dilated and ninety percent effaced, the fetal head was still ballotable, there were late decelerations and no accelerations, and the electronic fetal monitoring strip was Category II. (Klamerus Dep. 18:3-19:15.)

155. Dr. Klamerus's 7:22 a.m. note which she signed at 8:41 a.m. indicates that Mrs. Gharbi was off Pitocin and the electronic fetal monitoring strip was a Category II. (Klamerus Dep. 21:13-22:8.)

156. Dr. Klamerus verified that the note also documented that variability was absent, Mrs. Gharbi had late decelerations, her contractions were irregular at every four to seven

minutes, and her cervical change was only one centimeter, i.e., she was dilated to five centimeters, she was 100% effaced, and the fetal head was still ballotable. (Klamerus Dep. 22:18-24:4.)

157. Regarding the December 17th 8:00 a.m. sign out to Dr. Francis, Dr. Klamerus testified that stopping induction because of late decelerations was of clinical significance, stopping Pitocin because of late decelerations would have been relayed to Dr. Francis, the fact that Mrs. Gharbi was off Pitocin at sign out also would have been relayed to Dr. Francis, and Dr. Francis would have been made aware that Mrs. Gharbi had Category II strips. (Klamerus Dep. 31:14-32:5, 34:4-8.)

158. Dr. Klamerus confirmed that a cesarean section was not indicated at any time she was caring for Mrs. Gharbi. (Klamerus Dep. 36:15-17.)

159. After sign out, Dr. Klamerus was not involved in Mrs. Gharbi's care. (Klamerus Dep. 35:22-24.)

- **TESTIMONY OF HATTAB (BEN) GHARBI**

160. Plaintiff Hattab (Ben) Gharbi, the father of E.G., testified that he went to all of Mrs. Gharbi's prenatal visits and they had good reports at each visit except for one time when they were told there was excessive amniotic fluid that was not dangerous but would be monitored. (TT Day 2, 154:17-156:24.)

161. He said that they specifically asked about the amniotic fluid at their next visit, were told that everything was back to normal and heard nothing more about the problem or

had any other report that something was wrong. (TT Day 2, 156:25-157:12.)

162. Mr. Gharbi testified that he was with his wife at the hospital except when he drove his father home at some time on Sunday, December 17th. (TT Day 2, 12-17.)

163. He was not there when Dr. Francis ruptured Mrs. Gharbi's membranes at 3:00 p.m. (TT Day 2, 158:23-159:7.)

164. Mr. Gharbi testified that he did not meet Dr. Francis on the morning of the 17th--the first time he met her was that evening at about 7:30 when people rushed into the room and, in response to his question about what was going on, he was told that the baby's heartbeat went down. (TT Day 2, 159:11-19.)

165. He described what happened next:

> [T]hey started working on her.
>
> They were trying to read the heartbeat, actually, what they were trying to do, they had, like, two metal things, like, maybe a foot long, kind of . . . twisted wire, two of them they were trying to get them through her vagina, in order to read the baby's heartbeat. That was the first doctor.
>
> Second one, third one, and they couldn't succeed to do that. That's when I noticed that there was a person standing by the door, I mean, not even with the doctors, she was by the door -- I'm talking about Dr. Francis -- and she was standing and leaning on the door and just watching. When nobody succeeded to do that, that's when she jumped in and she tried to do that, and she pushed them in a little bit, and they still not reading anything.
>
> That's when she asked her, she asked my wife to flip over on her hands and knees, and that's when they got to read the baby's heartbeat, and after that, they say, well, now, you can go back to the other position, like, laying on back, and they left the room.

(TT Day 2, 159:19-160:13.)

166. Mr. Gharbi testified that he had called the nurses on the phone several times before the 7:30 event when the machine connected to Mrs. Gharbi's arm was beeping. (TT Day 2, 161:1-5.)

167. Each time, a nurse would come in, check the wires, check the fluid, fix it, push some buttons, and leave. (TT Day 2, 161:9-10.)

168. He said the next time he saw Dr. Francis was when she came in and told them they had to do an emergency C-section. (TT Day 2, 161:11-14.)

169. Mr. Gharbi was in the operating room seated next to his wife for the cesarean section and said that he stood up when the operating table started shaking and he saw that the baby "was completely dark" and "not responsive, I didn't hear him crying, I didn't hear him gasp or didn't see him moving, at all, period." (TT Day 2, 162:5-163:12.)

170. He further testified that as Dr. Francis was holding the baby and moved away from the operating table, the baby's head was falling back and his arms and legs were flopping away from his body. (TT Day 2, 163:13-18.)

171. Mr. Gharbi said that there was only one nurse in the room and, after Dr. Francis motioned to her, the nurse made some phone calls, Dr. Francis put the baby on a table, the baby's eyes were half open with only white visible, and then others came in and starting working on the baby but he did not respond. (TT Day 2, 163:20-164:8.)

172. Thereafter, one of those who had worked on the baby signaled to Mr. Gharbi that the baby was gone and then he was asked whether he wanted to cremate the baby or

bury him. (TT Day 2, 164:10-15.)

173. Mr. Gharbi testified that he never saw Dr. Francis again but one of the nurses expressed condolences and gave a photograph of the baby to his wife and they also got copies of the baby's footprints. (TT Day 2, 165: 3-25 (Joint Exhibits 9 and 11).)

174. He also testified that, while he and his wife were still in the room, Gregory Bowser (identified by Mr. Gharbi as Coroner or Deputy Coroner) walked in and said he was there because of their son's death. (TT Day 2, 167:14-16.)

175. Mr. Gharbi said that he wanted to do an autopsy to find out why his son died but Mr. Bowser refused and then said it would cost Mr. Gharbi a lot of money, estimating $10,000, and finally said it had to be done through the hospital. (TT Day 2, 167:18-168:11.)

176. He further testified that Mr. Bowser called him a few months later to tell him that his son died of Hirschsprung's Disease. (TT Day 2, 169:6-12.)

177. Mr. Gharbi confirmed that he, his wife, and the baby all had genetic testing done, all had normal karyotypes, and no further genetic testing was recommended. (TT Day 2, 170:1-171:14.)

- **TESTIMONY OF IMEN GHARBI**

178. Imen Gharbi, the mother of E.G., testified that all pretnatal visits were normal and, after the one instance of increased amniotic fluid, all ultrasounds were normal. (TT Day 2, 175:21-176:7.)

179. She testified that the first time she saw Dr. Francis was about 3:00 p.m. on

Sunday, December 17th at which time Dr. Francis did not introduce herself but said she was going to check Mrs. Gharbi's cervix. (TT Day 2, 177:6-9.)

180. Mrs. Gharbi said her water broke in the process. (TT Day 2, 177:13-14.)

181. Regarding the 7:30 p.m. event, Mrs. Gharbi said the residents and others came to her room all of a sudden, told her the baby's heart rate was going down, the first doctor's attempt to introduce wires vaginally did not succeed, but Dr. Francis stepped in and successfully attached the wires on her second attempt after she had Mrs. Gharbi flip to her hands and knees. (TT Day 2, 178:16-179:8.)

182. Mrs. Gharbi said when she asked Dr. Francis what caused the baby's heart rate to go down, Dr. Francis just looked at her and left the room, after which she did not see Dr. Francis until Dr. Francis told her she needed to do an emergency C-section at about 1:30 a.m. (TT Day 2, 179:18-180:4.)

183. When asked what she remembered about the C-section, Mrs. Gharbi said she kept waiting to hear the baby and didn't hear anything then kept telling her husband she wanted to see the baby but she did not see anything. (TT Day 2, 180:7-19.)

184. She said a nurse ultimately told her the baby had died. (TT Day 2, 181:3-5.)

185. Mrs. Gharbi confirmed that an autopsy had been done and that the coroner had called and told them that the cause of death was Hirschsprung's Disease, and that the genetic testing of she, her husband, and the baby all showed a normal karyotype. (TT Day 2, 181:15-182:14.)

- **TESTIMONY OF BROOKE CRUMMEL**

186. Brooke Crummel, an employee of UPMC Pinnacle who works in the Respiratory Department of the Harrisburg Hospital, testified remotely. (TT Day 3, 36:1-19.)

187. As the System Blood Gas Manager (since 2020), her daily work involves dealing with lab results at the facility. (TT Day 3, 36:24-37:3.)

188. Specifically, Ms. Crummel monitors the flow of results from point of care machines to the physician's charts and she deals with policies and procedures and competencies of staff members. (TT Day 3, 37:3-9.)

189. She testified that she is familiar with the format and terminology that the Pinnacle Lab uses when it records lab results in records. (TT Day 3, 37:18-21.)

190. When asked to look at lab results pertinent to the case, i.e., results which included analysis of the venous blood sample, Ms. Crummel was asked to explain terms used. (TT Day 3, 37:23-40:6.) She stated that "Lab results in on 12/18/17, 0241" means that the lab was collected on the blood gas analyzer; "Collected on" means the time that the blood sample was injected into the point of care machine which is the same as the "Lab results in on" activity; "Resulted:12/18/17, 0245. Result status: Final result" means the time that the results were viewable in that patient's chart; under the headings of "Component," "Comments," and "Value," "DT 0141" means the time that the blood was drawn; and "DT" stands for Draw Time, the time the specimen was drawn from the umbilical cord. (TT Day 3, 38:2-39:20.) Ms. Crummel further explained that the "DT" information comes from the labor

and delivery nurse collecting the sample and the importance of knowing the time is that the hospital has a policy that allows two hours to run the sample. (TT Day 3, 39:21-40:5.) Ms. Crummel also confirmed that, in this case, the report indicates that the draw time was 1:41 and the final results were available at 2:45. (TT Day 3, 40:10-14.)

191. Ms. Crummel had no involvement with the collection, processing or interpretation of Mrs. Gharbi's blood gas sample in December 2017 and she did not know whether the time 1:41 indicated as "DT" on Mrs. Gharbi's sample was referencing the baby's delivery time or whether the appropriate protocol was used. (TT Day 3, 41:3-42:3.)

192. Although Ms. Crummel acknowledged that she was not involved in the respiratory lab in December 2017, she confirmed that she was a respiratory therapist handling blood gases at the time and the format of the records and policies were the same as they had been in 2017. (TT Day 3, 42:8-43:23.)

193. She also confirmed that the lab record at issue and those like it are prepared in the ordinary course of business of the lab, are prepared contemporaneously with the events and the results that they record, are kept as records of what the lab performed and what the lab resulted on various samples, and that staff can and do make mistakes. (TT Day 3, 43:24-44:15.)

D. EXPERT TESTIMONY

- **JILL MAULDIN, M.D.**

194. Jill Mauldin, M.D., is a Maternal Fetal Medicine Specialist offered by Plaintiffs and accepted as an expert at trial in the fields of Obstetrics and Gynecology, Maternal Fetal Medicine, and Electronic Fetal Monitoring. (TT Day 1, 137:24-138:4.)

195. Dr. Mauldin explained that Maternal Fetal Medicine is a specialty that takes care of pregnancies complicated by maternal disease or fetal complications and she had training and certification with interpreting and reading electronic fetal monitoring strips. (TT Day 1, 128:17-129:3.)

196. She confirmed that she last delivered a baby in 2015. (TT Day 1, 204:15-17.)

197. She testified that her review of the prenatal records showed an uncomplicated pregnancy with no indication that the fetus had any difficulty swallowing amniotic fluid before delivery. (TT Day 1, 144:7-23.)

198. Dr. Mauldin further explained that an ultrasound at 36 weeks had increased fluid but before and after that the ultrasounds all had normal fluid, and the ultrasounds and evaluations always appeared normal, indicating that the baby was growing appropriately. (TT Day 1, 144:23-145:9.)

199. She posited that the OB-GYN notation related to the increased fluid, i.e., "[l]imitations of ultrasound," was essentially a normal finding not indicative of any kind of condition requiring treatment like polyhydramnios. (TT Day 1, 146:7-11.)

200. Dr. Mauldin agreed that the standard of care requires an OB-GYN to closely and actively monitor the baby's heart rate to make sure the baby is being adequately oxygenated, and to recognize in utero fetal signs evident on fetal monitoring strips and act on them by delivering the child before the effects of asphyxia, hypoxia, or acid base imbalance. (TT Day 1, 150:23-151:6.)

201. Dr. Mauldin opined that when a fetus is exposed to insufficient oxygen ("as they become less and less well oxygenated"), the variability and their heart rate pattern is going to become flatter. They typically will have decelerations in the heart rate that occur after the contractions, but as the babies become more and more sick, those decelerations can often become very subtle, and perhaps more difficult to pick up on. (TT Day 1, 152:23-153:8.)

202. Dr. Mauldin opined that she did not think that a fetus could withstand asphyxia, hypoxia, acidosis, or imbalance indefinitely: "there are always short periods of time that a healthy baby is going to be exposed to it and can tolerate it, but when it goes on for long periods of time, then we have to be more concerned that the baby [is] really being damaged, not being able to recover" and a fetus that is exposed in utero to the effects of asphyxia, hypoxia and acidosis is at an increased risk of death if not timely delivered. (TT Day 1, 151:25-152:13.)

203. Dr. Mauldin testified that if there's minimum heart rate variability persisting for hours accompanied with late decelerations and other decelerations it is an opportunity for continued hypoxia, and, in the setting of hypoxia, the baby becomes acidotic which can

result in some sort of neurologic damage or even death, and she agreed that persistent minimum variability and decelerations would be indicative of acidotic pH. (TT Day 1, 154:24-155:15.)

204. Dr. Mauldin testified regarding Category II strips that a small degree of decelerations can be allowed but if they are persisting for more than an hour or if the variability is gradually changing over an hour "we need to go to delivery." (TT Day 1, 156:17-24.)

205. She said that Mrs. Gharbi came in with a Category II strip and continued to have Category II strips through the administration of Cytotec and the start and discontinuance of Pitocin. (TT Day 1, 157:14-160:18.)

206. Dr. Mauldin said the standard of care was close monitoring of Mrs. Gharbi: it did not allow Dr. Francis to transfer the responsibilities regarding Mrs. Gharbi's labor course to her residents; it was below the standard of care for Dr. Francis to have assumed care of Mrs. Gharbi at 8:00 a.m. on December 17th and a resident (Dr. Lin) not to see Mrs. Gharbi until five hours later; and Mrs. Gharbi should have been evaluated within an hour of the team coming on at 8:00. (TT Day 1, 160:20-162:25.)

207. When asked whether Dr. Francis had an obligation on the morning of December 17th to make a decision concerning the course of Mrs. Gharbi's labor management, once she assumed care, after having been advised by Doctors Klamerus and Maenner that they tried Pitocin had to be stopped because of late decels and so forth (TT

Day 1, 163:5-9), Dr. Mauldin responded as follows:

> She definitely -- I mean, it turned over to her responsibility. I don't know that the baby had to be delivered by C-section right then that morning at 8 or 9, I know, in her deposition, she said she would have done a C-section then. For sure, the patient needed to be evaluated and thoroughly assessed and a decision point made, you know, Do we keep trying to go vaginally for delivery? You know, Can we use Pitocin again and get farther? Or is a C-section indicated?

(TT Day 1, 163:10-17.)

208. Dr Mauldin found it reasonable to restart Pitocin at approximately 2:30 p.m. on December 17, 2017, since the tracings did not show frequent decelerations at that point and they needed to have contractions to get delivered but Dr. Francis's lack of awareness of the stopping and starting of Pitocin was an example of Dr. Francis's failure to actively and closely monitor Mrs. Gharbi's labor course. (TT Day 1, 164:23-165:22.)

209. Dr. Mauldin found that Dr. Francis acted appropriately when she ruptured Mrs. Gharbi's membranes at 3:00 p.m. (TT Day 1, 167:6-8)

210. Dr. Mauldin stated that Mrs. Gharbi had an arrest of labor which means she had no change in her cervical exam over a period of four hours—Mrs. Gharbi's cervix remained at seven centimeters dilated from 3:00 p.m. to 7:00 p.m. on December 17th which was a very appropriate reason to do a cesarean section and Dr. Francis's failure to do so breached the standard of care. (TT Day 1, 167:15-168:12.)

211. Dr. Mauldin added that a decision to do a cesarean was also appropriate at 7:00 p.m. because it was clear much earlier in the labor process that there was a dysfunctional labor curve and an abnormal labor pattern with a fetus that has had a

Category II strip and has had periods of decelerations or periods of hypoxia—"there are too many things that are just warning signs that this is not normal." (TT Day 1, 168:15-22.)

212. Dr. Mauldin opined that the failure to deliver at 7:00 increased the risk of harm to the fetus in that the death could have been prevented if delivery had occurred at the 7 p.m. arrest of labor--the "14-minute deceleration" or "prolonged deceleration" that happened at 7:24 p.m. and the subsequent hour-long difficulty evaluating the fetal heart rate because of some artifact on the machine would not have occurred. (TT Day 1, 170:12-171:8.)

213. Regarding the 7:24 p.m. prolonged deceleration, Dr. Mauldin agreed that the records showed a "14-minute deceleration" which, by definition, was an acute bradycardic event that falls into a Category III strip and provides another reason that the baby should have been delivered and Dr. Francis's failure to do so was below the standard of care and increased the risk of harm and death to the baby. (TT Day 1, 171:12-172:20.)

214. Dr. Mauldin did not state in any one of her four reports or in her deposition that the tracings were ever a Category III. (TT Day 1, 223:10-13.)

215. At trial, Dr. Mauldin agreed that she "interpreted the tracings in this case as the majority of labor had a Category II tracing" at her deposition and, in her November 22, 2020, report she described the tracings as "unrelenting Category II fetal monitoring." (TT Day 1, 221:23-222:4.)

216. Dr. Mauldin provided a detailed assessment of the electronic fetal monitoring strips related to the period she referred to as the prolonged deceleration which she said

indicated the fetus was not getting adequate oxygenation, exposing the fetus to asphyxia, hypoxia, and acid base imbalance:

- During the period, the heart rate went from the normal range (120 to 160) into the 80's;
- There is a period of forty seconds where the heart rate is remaining in the 140's but then it goes down again to the 90's;
- There are periods when the heart rate cannot be seen at all;
- There was a period of nine minutes with no traceable heart rate in the normal range;
- There was a slow return to baseline where the heart rate was coming from below 90 and five minutes later it was up to the normal range of 100 or greater;
- There is also a period where, due to monitor interference or some other reason, there is not a good tracing and you don't know what's going on with the baby but you would expect that, after a prolonged deceleration, the baby would have problems for another twenty to thirty minutes. (TT Day 1, 173:5-178:5.)

217. Dr. Mauldin described the 14-minute period beginning at 7:24 p.m. (during which she says there was never an adequate return to baseline) as a "sentinel event" by which she meant that "we have had a catastrophic and really unexpected outcome." (TT Day 1, 178:8-22.)

218. At trial, Dr. Mauldin confirmed that she stated at her deposition that there was a twelve-minute period beginning at 7:29 p.m. on December 17th that the monitor was unable

to capture the fetus's heart rate, stating "[i]t is probably a mechanical function that, for whatever reason, we're not picking up the heart rate. Is the heart rate down too low? Has the baby moved positions? But certainly it's concerning, because the minutes before the loss of continuous heart tracing, the baby was showing signs of distress." (TT Day 1, 229:17-22.)

219. Dr. Mauldin agreed that the 14-minute period which she considered the sentinel event includes twelve minutes where the monitor was not recording the fetus's heart rate and forty or fifty seconds of the heart rate returning to baseline. (TT Day 1, 234:9-235:18.)

220. Although Dr. Mauldin concurred that, during this period, the fetus's heart rate could not be known because the monitor was not able to capture it, she stated that she believed that, because the heart rate was going down before the malfunction, it should have been assumed that the baby was likely in distress, an assumption that is in the best interest of the patient and is supported by a rate in the 60's and 70's when the tracing returned and the fact that it took five minutes or more for the rate to return to baseline. (TT Day 1, 229:25-234:20.)

221. Dr. Mauldin later stated that, rather than a mechanical malfunction, she believed that the rate was not being picked up during this period because the heart rate was very low and the monitor often does not pick up a rate that low. (TT Day 1, 248:25-249:10.)

222. Dr. Mauldin followed this assessment with the confirmation that she believed

there was evidence of hypoxia and acidemia at this time which could neither be seen on autopsy nor shown in the core blood results. (TT Day 1, 249:13-18.)

223. She stated that "[t]ypically, most obstetricians, I mean, we don't have something in the literature, but usually after a baby—after we have been in a deceleration for 8 definitely 10 minutes, then, we just move to delivery at that point by stat C-section." (TT Day 1, 177:3-6.)

224. Dr. Mauldin did not believe that the return of the heart rate to baseline meant that everything was okay in that the tracings continued to show minimal variability and the occurrence of late decelerations. (TT Day 1, 178:23-179:4.)

225. Dr. Mauldin stated that the tracings from 7:53 p.m. to 8:09 p.m. (Exs. J-3.208, J-3.209) continued to be attributable to some artifact on the machine (machine not tracing appropriately) so she thought it could not be assessed whether there was any subtle late deceleration after each contraction which is what she would have expected. (TT Day 1, 181:15-182:1.)

226. Beginning at 8:10 p.m. (Ex. J-3.210), Dr. Mauldin stated that it looked like the tracing was improving and she saw a late deceleration, followed by absent variability and very subtle decelerations in the following nine-minute period. (Ex. J-3.211). (TT Day 1, 182:2- 183:2.)

227. Dr. Mauldin opined that the time period ending at 8:29 p.m. (Ex. J-3.211) indicated that the fetus had lost more of its reserves "as it was going from minimal variability

down to absent variability after that prolonged decel, . . . we understand that to mean that the baby is losing or, perhaps, has even lost all of its oxygen reserves." (TT Day 1, 183:8-23.)

228. She also opined to a reasonable degree of medical certainty that the strip ending at 8:29 p.m. continued to evidence that the fetus was suffering from the effects of asphyxia, hypoxia and acidosis. (TT Day 1, 184:10-16.)

229. Dr. Mauldin interpreted the following strip (Ex. J-3.212) to have minimal variability with a late deceleration which she found to be continued evidence that the fetus was hypoxic and acidotic. (TT Day 1, 184:17-185:2.)

230. Dr. Mauldin continued to assess minimal to absent variability with subtle decelerations over the next two time periods which ended at 8:53 p.m. (Ex. J-3.213, J-3.214) and opined to a reasonable degree of medical certainty that the baby should have been delivered because it was showing evidence that it was not doing well. (TT Day 1, 185:6-187:3.)

231. Dr. Mauldin confirmed that after 9:00 p.m. she found late decelerations in over half of Mrs. Gharbi's contractions which was evidence of hypoxia and acidosis jeopardizing the baby's health. (TT Day 1, 187:4-20.)

232. Dr. Mauldin opined that the strips from this time period and throughout the remaining duration of fetal monitoring evidence that the fetus was suffering from the effects of asphyxia, hypoxia, and acidosis; they indicate the baby should have been delivered; they

show that Dr. Francis should have been making an action plan to deliver the fetus; and they show that her failure to deliver increased the risk that the baby would suffer adverse effects from ongoing hypoxia, asphyxia and acidosis. (TT Day 1, 188:6-190:11.)

233. Dr. Mauldin agreed that her general concept for Mrs. Gharbi's labor, from admittance to the hospital to delivery, was that there was minimal variability and there were recurrent decelerations through the entire labor. (TT Day 1, 222:17-223:1.)

234. Based on her finding that there was "very clear evidence that there was fetal distress happening in [the] six hours prior to delivery and . . . a very guarded course since admission," Dr. Mauldin disagreed with Dr. Christian Pettker's assessment that there was no fetal distress in the electronic fetal monitoring at the time proximal to birth, that there was no evidence of hypoxia, and that the strips in the hour before delivery did not show any abnormality that would indicate the fetus was at risk of death at birth. (TT Day 1, 191:5-193:2.)

235. Dr. Mauldin agreed that there can be differences in the interpretations of tracings but she "didn't think that there should be differences . . .as the situation becomes worse." (TT Day 1, 217:10-218:3.)

236. Dr. Mauldin concluded that the cause of Baby Gharbi's death was the cumulative effects of hypoxia and acidosis and the death was preventable had the baby been delivered no later than 9:00 p.m. on December 17, 2017. (TT Day 1, 193:9-20.)

237. The "signs" Dr. Mauldin relied on to conclude that death was caused by

hypoxia and acidosis are the fetal heart rate tracings. (TT Day 1, 238:2-6.)

238. On cross-examination, Dr. Mauldin agreed that, at her deposition, she said she was offering her "best guesstimate" because she was not an authority on diseases or what happens with a neonate, and, on redirect, she stated that her opinions regarding the breach of the standard of care and cause of death were based on a reasonable degree of medical certainty. (TT Day 1, 257:23-258:14.)

239. Dr. Mauldin agreed that a baby born hypoxic is not necessarily harmed and a baby can be born with hypoxic injury and acidosis and survive. (TT Day 1, 238:12-17.)

240. Dr. Mauldin agreed that lab results showed a pH value of 7.353, that value collected from a double-clamped sample of cord within one minute after delivery would be a normal pH value for a neonate, and cord gas results would not become more normal if they were drawn too late. (TT Day 1, 243:1-6, 249:19-21.)

241. She also agreed that her opinion expressed at her deposition that the cord gas results were unreliable was based on her belief that the sample was collected one hour after delivery. (TT Day 1, 244:9-17.)

242. Despite information that the cord gas sample was taken right after delivery, Dr. Mauldin believed that the normal cord gas result did not fit the clinical picture. (TT Day 1, 246:18-247:19.)

243. She further expressed that, if she were to assume the venous cord blood was timely collected, her opinion, to a reasonable degree of medical certainty, would not change

in any way regarding the baby's death from hypoxic, asphyxic and acidotic injury and death. (TT Day 1, 197:5-19.)

244. Dr. Mauldin stated that she did not believe that a single normal venous blood gas could determine whether a baby has suffered from asphyxia, hypoxia, or acidosis because, in addition to not knowing when the sample was drawn,

> it looks like it was not reported for a whole hour after the baby was born, and the test itself, I think, has a fairly quick turn-around, so I think just looking at a single item, you know, we have to look at the whole clinical course and know that the things are not matching. You know, so we have many more variables that are consistent with the death that occurred because of hypoxia and acidosis rather than this one blood gas value.

(TT Day 1, 195:24-196:14.)

245. Regarding the testing of cord gases after delivery, Dr. Mauldin stated that the testing indicated the metabolic and respiratory status at the time of delivery, a sampling of arterial blood would be important because the sampling from the umbilical vein would typically be in the higher or more normal appearing range than the umbilical artery, the timing of the collection of the cord blood gas can skew results, and proper clamping and timing are important for reliability. (TT Day 1, 194:3-195:4.)

246. Confirming that a cord can be either single clamped or double clamped and that Dr. Francis did not document what she did, Dr. Mauldin explained that an umbilical cord is attached to the placenta after it has been delivered so there is no risk of the mother bleeding, and, if the cord is single clamped, she agreed that the sample can be open to the air which might have an impact on the results of the cord gas. (TT Day 1, 195:5-23.)

247. Dr. Mauldin agreed with Plaintiffs' counsel that a normal venous cord value could not exclude acute intrapartum asphyxia and you could have a normal venous cord value with fetal cardiac arrest and death. (TT Day 4, 196:15-20.)

248. As to the latter, Dr. Mauldin explained that

> under unusual circumstances, you know, it's rare but very possible that . . . a baby can . . . have a cardiac arrest in that very brief period of time between taking the mom off the monitor and getting the baby delivered, which, depending on how fast the procedure goes, could be a minute or it might even take five minutes . . . depending on the situation. So, . . . if the baby had a cardia arrest in that period of time, then, very possibly, that blood gas result hasn't had time to equilibrate.

(TT Day 1, 196:20-197:4.)

249. Dr. Mauldin concluded that the delivery record's documentation that the baby was pale and unresponsive, didn't cry, didn't have respiratory efforts, his pupils were non-responsive and arms and legs were contracted were clinical features consistent with death from asphyxia, hypoxia and acidosis during prolonged labor. (TT Day 1, 198:21-199:1.)

250. Regarding the contractures, she stated that contractures could be seen on an ultrasound and she saw no evidence of the fetus having contractures during the prenatal course. (TT Day 1, 145:16-22.)

251. She also concluded that the baby's lack of response to CPR was evidence of severe hypoxia and acidosis that could not be corrected, a baby can have contractures just from hypoxia, and there was no indication of any congenital condition causing his death. (TT Day 1, 198:18-200:23.)

252. On cross-examination, Dr. Mauldin agreed that she had no opinion on why E.G. was unable to be resuscitated. (TT Day 1, 250:1-3.)

253. Dr. Mauldin stated that it was unlikely that a genetic disorder caused E.G.'s death. (TT Day 1, 250:8-13.)

254. On cross examination, Dr. Mauldin stated that she did not have an opinion about the limb contractures reported in the record to be apparent upon delivery but she stated that there was no evidence of contractures on prenatal care records or ultrasound. (TT Day 1, 251:24-252:15.)

255. Dr. Mauldin also testified on cross-examination that the description of doughy skin in the record had no impact on her opinions and she had no explanation for the non-floating lungs at autopsy. (TT Day 1, 243:1-6.)

256. Dr. Mauldin disagreed with Dr. Pettker's assessment that the absence of hypoxic injury on the pathology report indicates that the baby didn't suffer hypoxia or acidosis significant enough to cause organ or tissue damage at birth because she thought there was not enough time for that to develop in that it usually takes twenty-four to forty-eight hours to develop. (TT Day 1, 201:11-22.)

257. On cross-examination, Dr. Mauldin confirmed that she was "opining on the cause of death of the neonate, with reference only to the fetal heart rate tracings in utero and not with reference to the APGAR scores, the inability to resuscitate, the possible genetic disorder, a record of doughy skin, limb contractures, possible Hirschsprung's

Disease, abnormal cells, non-floating lungs, cord blood gas results or possible undiagnosed neurological issue." (TT Day 1, 256:10-17.)

258. To summarize, on direct examination, Dr. Mauldin opined, to a reasonable degree of medical certainty, that (1) E.G. died from hypoxic acidotic injury during protracted labor, (2) the death was preventable, (3) when the strip converted to a Category III, Dr. Francis was required by the standard of care to deliver by cesarean, and (4) the additional six hours she waited to deliver the baby increased the risk that he would die from asphyxia, hypoxia, and acidosis. (TT Day 1, 197:23-198:13.) On cross examination, Dr. Mauldin was asked to confirm the opinions she had expressed on direct examination: Dr. Mauldin's opinion on cause of death was that E.G. died from hypoxic acidotic injury that occurred during labor, that he died from acidosis, and that he had hypoxic episodes during labor that led to the acidosis (TT Day 1, 203:20-204:7); Dr. Mauldin opined that E.G. died shortly after birth from hypoxic and acidotic injury due to protracted labor and failure to deliver by C-section (TT Day 1, 204:8-11); Dr. Mauldin opined that acidosis was the sole cause of death (TT Day 1, 204:12-14); Dr. Mauldin opined that Dr. Francis fell below the standard of care when she did not deliver by cesarean with the arrest of labor at 7:00 p.m. on December 17th (TT Day 1, 210:15-19); and Dr. Mauldin opined that Dr. Francis fell below the standard of care when she did not deliver the baby by cesarean after the event which began at 7:24 p.m. on December 17th (TT Day 1, 211:5-212:6).

- **TESTIMONY OF JENNIFER HAMMERS, D.O.**

259. Jennifer Hammers, D.O., is a Forensic Pathologist offered by Plaintiffs and accepted as an expert in the fields of Clinical, Anatomic, and Forensic Pathology. (TT Day 2, 12:7-12.)

260. Based on her review of the records, Dr. Hammers opined to a reasonable degree of medical certainty that Baby Gharbi died from an intrauterine fetal demise due to hypoxia and acidosis following prolonged labor and delivery. (TT Day 2, 16:8-11.)

261. Dr. Hammers did not offer an opinion on the standard of care. (TT Day 2, 57:2-4.)

262. Dr. Hammers does not deliver babies, she is not an OB-GYN practitioner, and does not read fetal heart monitoring strips as part of her daily work. (TT Day 2, 57:5-13.)

263. Dr. Hammers confirmed that she is not an expert at reading fetal heart tracings, that hypoxic injury is not the only reason a baby can have a low APGAR score, and blood tests are more objective than APGAR scores. (TT Day 2, 84:1-85:2.)

264. She testified that she would leave the reading of fetal heart rate strips to other experts. (TT Day 2, 84:1-6.)

265. Dr. Hammers found Mrs. Gharbi's prenatal course significant for its uneventfulness—there was nothing abnormal in her prenatal course except for one instance in November of increased fluid around the baby, polyhydramnios, which was not present on the previous or subsequent ultrasounds. (TT Day 2, 16:16-17:14.)

266. Dr. Hammers disagreed with Dr. Pettker's opinion that Mrs. Gharbi's pregnancy was complicated by polyhydramnios, stating that the finding of polyhydramnios on one ultrasound that resolved itself does not indicate any bigger or persistent issue with the baby in that a bigger issue would have been persistent or continually showing concerning levels of amniotic fluid. (TT Day 2, 17:15-18:4.)

267. Dr. Hammers found Mrs. Gharbi's labor course significant because of documented episodes of changes in the fetal heart monitoring strips that were concerning enough for physicians to treat Mrs. Gharbi—giving her fluids and having her change positions—and changing their treatment of her to progress through labor. (TT Day 2, 19:9-25.)

268. Regarding the period referred to as the 14-minute deceleration, Dr. Hammers said that the decelerations and fetal heart tracings indicated that there was likely some other process going on related to the fetus not getting enough oxygen--the tracings showed that the fetus was experiencing stress that involves the amount of oxygen the fetus is getting and decreases in oxygen can lead to acidosis which is a rearrangement of the balance of oxygen, hydrogen, and carbon dioxide in the baby's body. (TT Day 2, 20:4-15.)

269. Dr. Hammers opined that, to a reasonable degree of medical certainty, E.G. had a critical level of decreased oxygen that resulted in his death: the hypoxia during the prolonged labor and delivery was the reason that he was born with an APGAR of zero and why he was not able to be successfully resuscitated. (TT Day 2, 21:6-9.)

270. She explained that you can have death related to a decrease in oxygen either very suddenly or over the course of a period of time: if oxygen is completely cut off to the body, the body has some oxygen reserves that it can utilize before suffering the consequences of not having oxygen; if decreased levels of oxygen occur over time, the body depletes its resources, little by little, until it reaches a critical level where damage begins to occur. (TT Day 2, 21:13-20, 56:6-10.)

271. Dr. Hammers stated that the prolonged induction and the decelerations and minimal variability caused E.G. to have a critical low level of oxygen that resulted in his death. (TT Day 2, 22:1-5.)

272. She explained that "looking at the clinical records and the course of what happened with this baby and ruling out other causes that were significant that could cause this baby's death, when you put the whole picture together, we're left with the baby having hypoxia related to this prolonged period of labor and delivery." (TT Day 2, 31:10-15.)

273. Dr. Hammers opined to a reasonable degree of medical certainty that the six-hour period of time between the 14-minute deceleration at around 7:30 p.m. and the 1:41 a.m. delivery impacted the baby's ability to be successfully resuscitated from the hypoxic injury he suffered. (TT Day 2, 29:4-12.)

274. Dr. Hammers stated that the brain is the organ of the body most affected when a fetus is exposed to a hypoxic and acidotic environment and the brain feeds back to the heart through the brain stem so as the brain is affected, the brain stem becomes affected

and the brain stem is what tells the heart to beat so changes in the brain will directly affect the heart because the brain is not able to send those signals to keep it beating normally and effectively. (TT Day 2, 32:15-25.)

275. Regarding the lack of evidence of hypoxic death at autopsy, Dr. Hammers stated she would not expect to see any changes in the brain under the microscope because it takes time for the body to respond to the damage and mount a response and here there were no identified changes in the brain because a process of a few hours doesn't allow the body to respond—the decreased oxygen occurred over time and the body was able to handle those decreases over time until it reached a critical level and, once it reached a critical level, causing the baby to die, the body did not live long enough to mount a reaction where changes would show under a microscope. (TT Day 2, 30:4-31:9.)

276. Dr. Hammers stated that the fact that a neonatologist was called to the delivery meant that it was not a normal course of delivery. (TT Day 2, 22:16-25.)

277. Dr. Hammers opined that E.G.'s death was intrauterine because it was clear when he was delivered that he was not responsive and his skin discoloration indicated that he was not receiving oxygen and that he was deceased. (TT Day 2, 23:6-17.)

278. She stated that the record's description of E.G. as pale, unresponsive, with no cry or respiratory effort and non-responsive pupils was consistent with death as a result of hypoxic and acidotic injury. (TT Day 2, 26:1-7.)

279. Dr. Hammers also stated that the record's indication that the baby was pale

with petechiae throughout the face, neck, upper torso, with poor profusion and hypothermic was consistent with hypoxic and acidotic injury because hypothermia can occur from the baby being deceased, the coloration of the baby and petechial hemorrhages identified on the skin indicated that there's some kind of increased pressure that's causing small blood vessels in the skin to break which is reflective of the prolonged period of time that the labor and delivery was going. (TT Day 2, 26:19-27:10.)

280. Dr. Hammers testified that she made note of the venous blood gas obtained after delivery but her opinion doesn't change whether the blood gas is valid or invalid. (TT Day 2, 33:1-24.)

281. Dr. Hammers agreed that objective measures of acidosis are a group of tests that are run to make the determination. (TT Day 2, 64:14-65:10.)

282. Although Dr. Hammers said, in general, the blood pH itself could indicate whether there was acidosis, she stated that, in the specific context of this case, there was acidosis even though she agreed that the pH value was in the reference range (having stated in her first report that clinical testing of the venous cord blood gas revealed acidosis and, in her supplemental report, that the lab values are not reliable in formulating opinions about acidosis in this case based, in part, on Dr. Francis's testimony about when the cord blood sample was collected, i.e., that there was a prolonged time between collection and testing). (TT Day 2, 68:2-5, 69:9-17, 75:22-76:23.)

283. Dr. Hammers stated that, in the postmortem setting, testing can be unreliable

and is not validated so, from her perspective as a Forensic Pathologist, "we do not run testing or initiate testing on postmortem samples for things like, electrolytes, blood gases, things like that, because they're not validated. So, from my perspective, if something is done in the postmortem period, then, I don't know how to interpret it." (TT Day 2, 80:13-19.)

284. She agreed that acidosis is not something that can typically be identified at autopsy, is not something a Forensic Pathologist tries to identify at autopsy, and umbilical cord blood gas analysis is not part of an autopsy. (TT Day 2, 91:21-92:2.)

285. Dr. Hammers confirmed that she disagreed with the Medical Examiner's and Coroner's Office listing of the cause of death. (TT Day 2, 59:17-20.)

286. Dr. Hammers discounted the death certificate completed by the deputy coroner listing complications of Hirschsprung's Disease and probable genetic disorder as the cause of death because it appeared to be based on the findings in the autopsy report and the autopsy report did not list the cause of death but rather contained focal findings that were not significant regarding a congenital syndrome and did not explain why E.G. died when he did under the circumstances presented. (TT Day 2, 34:5-35:13.)

287. Turning to the autopsy report and the recognition of congenital syndromes where both Hirschsprung's and limb contractures may be seen, Dr. Hammers explained the pathologist's assessment that the limb contractures would be expected to be much more pronounced if they were related to a congenital syndrome that would have been of significance. (TT Day 2, 35:24-36:8.)

288. Dr. Hammers also explained why Hirschsprung's Disease would not cause the baby's death. (TT Day 2, 36:20-37:13.)

289. Dr. Hammers disagreed with Dr. Polin's opinion that Hirschsprung's affected the entire GI tract, stating that there are focal areas of the GI tract that are involved but they are not described as large and the autopsy does not describe the affected areas as the majority of the GI tract. (TT Day 2, 39:10-19.)

290. Regarding contractures, Dr. Hammers opined that they did not play a significant role in E.G.'s death and they would not have caused sudden or unexpected death or contributed to the events that led to his death. (TT Day 2, 41:15-21.)

291. On the relationship of contractures and hypoxia death, Dr. Hammers stated that hypoxia is definitely a cause of contractures: the hypoxia affects the brain, the brain can affect the musculature and joints, not in the long term but in a more immediate fashion and here there was never any evidence on the ultrasounds and prenatal examinations that the fetus had contractures in utero, there was never any decreased movement that can be associated with contractures, there was no decrease of amniotic fluid, and the musculature was described as normal. (TT Day 2, 42:2-15.)

292. Based on the lack of evidence of contractures in utero, Dr. Hammers disagreed with Dr. Polin and Dr. Chung who suggested a long-standing neurologic condition. (TT Day 2, 44:2.)

293. Dr. Hammers disagreed with other experts who opined that the autopsy finding

that the lungs did not float could be indicative of a genetic disorder and believed that the non-floating lungs indicated that "the baby didn't have a significant inspiratory effort, it wasn't breathing when it was delivered, and it never breathed after it was born, so those lungs never opened up and never developed those air sacks, so that they wouldn't float." (TT Day 2, 50:13-17.)

294. Dr. Hammers opined to a reasonable degree of medical certainty that a congenital or genetic abnormality did not cause E.G.'s death. (TT Day 2, 47:4-12.)

- **TESTIMONY OF WENDY CHUNG, M.D.**

295. Wendy Chung, M.D., a Physician Scientist at Columbia University, testified remotely. (TT Day 3, 48:21-24.)

296. Dr. Chung was offered by Defendant and admitted as an expert in the field of Medical Genetics and Clinical Pediatric Genetics. (TT Day 3, 57:25-58:2.)

297. Dr. Chung opined that E.G. did not have hypoxia or acidosis contributing to his death but rather had "several congenital anomalies . . . likely associated with an undiagnosed genetic condition, which likely contributed to his death. (TT Day 3, 58:23-59:2.)

298. Dr. Chung testified that the basis for her belief that E.G. did not have acidosis developed in utero is that there was an umbilical cord blood gas that was taken immediately after delivery, and for that cord blood gas, the pH was within the normal range. (TT Day 3, 59:5-9.)

299. Regarding E.G.'s pH of 7.353, Dr. Chung's report stated that it did not indicate

significant acidosis and, when asked on cross-examination at trial to confirm that meant the baby had acidosis, she stated that "a normal pH is 7.4 and when you start rounding up to two digits, Baby Gharbi's pH would be 7.4 so he had "very, very minimal acidosis . . . [not] something that would have been associated with a lethal event . . . 6 point something [being] what we usually see when we have a severe acidosis problem." (TT Day 3, 80:24-82:7.)

300. When asked whether she based her opinion that E.G. did not die from hypoxia or acidosis purely on the venous cord blood gases, Dr. Chung responded, "that was the only data point that I had to look at acidosis." (TT Day 3, 81:18-22.)

301. Dr. Chung acknowledged the lack of an arterial sample and explained that "the correlation between venous and arterial is quite commonly highly associated" and the level in this case was "not anywhere near the range that we see when we have a severe acidosis problem." (TT Day 3, 82:3-7.)

302. As to a delay in collecting the cord blood sample and associated reliability, Dr. Chung stated that her understanding was that you would more likely have a false positive if the sample is not immediately processed. (TT Day 3, 82:11-19.)

303. Dr. Chung testified that she formed an opinion as to why E.G. died based on the autopsy that demonstrated several anomalies including Hirschsprung's Disease, pyloric stenosis, contractures, evidence of the lungs not floating or not inspiring, doughy feet or other things that would suggest dysmorphisms in terms of the way the body had formed in

several different areas, not just limited to one organ system but in several different organ systems. (TT Day 3, 59:10-21.)

304. Dr. Chung further explained that, when you see that many different things going on, and considering that there was a history of polyhydramnios, that there were likely problems in terms of the way the brain was functioning (for instance, when the baby was needing to inspire his first breaths) that led to problems that would cause issues postnatally. (TT Day 3, 59:24-60:9.)

305. She confirmed that she holds these opinions to a reasonable degree of medical certainty. (TT Day 3, 60:10-13.)

306. Dr. Chung later clarified that when she said the larger the number of anomalies, the greater the likelihood of genetic etiology, she did not consider polyhydramnios an anomaly. (TT Day 3, 84:10-16.)

307. Dr. Chung stated that genetic abnormalities can be de novo, meaning that it is not something passed down or was not inherited from parents or grandparents but rather started new in the individual affected. (TT Day 3, 62:21-63:1.)

308. Dr. Chung described her process in this case as follows: she reviewed hospital records around the delivery, reviewed the autopsy report that included microscopic histology to be able to see things like Hirschsprung's Disease, and reviewed genetic test results. (TT Day 3, 64:1-6.)

309. Regarding genetic testing, Dr. Chung stated that karyotype testing, which E.G.

and Mr. and Mrs. Gharbi had, looks at chromosomes, does not exclude the vast majority of genetic conditions, and most people with genetic conditions have normal karyotypes and the parents have normal karyotypes. (TT Day 3, 76:23-77:12.)

310. She also said that karyotype testing "is just the tip of the iceberg in terms of genetic testing that could be performed" and, although she would have done additional testing, it was not recommended by the pathologist or done in this case. (TT Day 3, 95:12-96:3.)

311. She explained that

> when you see someone that has a single structural congenital anomaly, we call that isolated. When we see individuals that have more than one part of the body that seems to be affected, we often call that complex . . . [in which case] it is very likely . . . there is one single thing causing what we call pleiotropic effects, that is, that seem to have a myriad of manifestations on multiple parts of the body.

(TT Day 3, 64:20-65:4.)

312. Regarding autopsy findings, Dr. Chung stated that limb contractures of any type, Hirschsprung's disease, pyloric stenosis, intestinal atresia and doughy skin are not seen in most newborns "so you start thinking this can't be chance alone and these things must be connected." (TT Day 3, 66:22-67:19.)

313. Dr. Chung said she had never seen hypoxia or acidosis in a newborn cause contractures. (TT Day 3, 68:22-25.)

314. Regarding polyhydramnios, Dr. Chung stated that it was not normal although it was not severe and did not require treatment. (TT Day 3, 70:7-18.)

315. Dr. Chung also explained why pyloric stenosis is not normal and impacted her decision based on it being part of a larger picture and also why the limb contractures, though not noted on prenatal ultrasounds, could have been missed prenatally and the fact that they were present postnatally impacted her decision. (TT Day 3, 70:19-72:2.)

316. Regarding Hirschsprung's Disease, Dr. Chung stated that it can be surgically treated, its presence was important to her opinion because it was part of the constellation of features that very strongly suggests that something very early in the development went wrong and, with numerous problems below the shoulders, likely there were problems above the shoulders in terms of brain function and development which could have impacted basic functions like breathing after birth. (TT Day 3, 72:16-74:3.)

317. On cross-examination, Dr. Chung clarified that she understood that the Autopsy reported "possible Hirschsprung's Disease" but that still indicated abnormality because the Autopsy found aganglionic segments consistent with possible Hirschsprung's Disease and aganglionic segments are not normal and are a developmental anomaly. (TT Day 3, 90:13-91:14.)

318. Regarding doughy skin, Dr. Chung agreed on cross-examination that the autopsy did not state that E.G. had doughy skin or abnormal footprint but doughy skin was noted in the medical record, often in children doughy skin is noticed in the feet and the footprint she reviewed showed a lack of creases, and doughy skin, on any part of the body, is abnormal. (TT Day 3, 98:18-101:15.)

319. In sum, Dr. Chung opined to a reasonable degree of medical certainty that there were multiple different things that must have happened early in development that affected multiple parts of the body, explaining that

> each one of these is individually very rare. You put the combination of these very rare things together, so problems like contractures, problems like Hirschsprung's Disease, problems like pyloric stenosis, all of those, even the puffy feet, each one of those are individually rare, and, now, if you think of the likelihood that each of them would have independently happened by chance, it becomes nearly impossible. It's very much more likely these are all related, in terms of one underlying problem. When we think of problems that are this, sort of, exhaustive, in terms of the effect upon different parts of the body, they almost always involve the brain, so they almost always have issues, in terms of how the brain functions. Even if when you just look at the brain, grossly, it looks like it has all the parts of the brain, from a functional point of view, the brain is rarely working normally. So there is some evidence that the brain, even starting in utero, was not working normally with things like the polyhydramnios, problems with the contracture, and then postnatally, problems, likely, in terms of inspiration or breathing or taking that first breath. So it's likely that, unfortunately, this baby just had a very, very severe problem that was affecting many, many different parts of the body and was, unfortunately, just a lethal condition.

(TT Day 3, 79:2-80:2.)

320. When asked on cross-examination whether she had any association with Richard Polin, M.D., a Columbia Neonatologist and expert in this case, and whether they had collaborated in the past, Dr. Chung said they had worked together for twenty years but had not collaborated and did not work together in preparing their reports or declarations in this case and had not discussed the case. (TT Day 3, 104:15-105:8.)

321. Dr. Chung stated that counsel had helped with the format of the Declaration and confirmed that she personally and independently prepared it. (TT Day 3, 105:16-18.)

322. When asked about the similarity of her Declaration and that of Dr. Polin as demonstrated by a side-by-side comparison of the documents, Dr. Chung said that, in some instances, they were both using common medical terminology and repeated facts of the case, some of which may have been provided by defense counsel. (TT Day 3, 107:1-109:10.)

- **CHRISTIAN PETTKER, M.D.**

323. Christian Pettker, M.D., a physician on the medical staff for Yale New Haven Hospital and Professor in the Department of Obstetrics, Gynecology and Reproductive Sciences in the School of Medicine at Yale, testified remotely. (TT Day 4, 3:21-4:11.)

324. He was offered by Defendant and accepted as an expert in the field of Maternal Fetal Medicine. (TT Day 4, 8:13-14, 11:25-12:1.)

325. Dr. Pettker opined to a reasonable degree of medical certainty that he did not believe E.G. was harmed by a lack of oxygen in utero during the labor process or was at risk for death because of a lack of oxygen. (TT Day 4, 13:6-11.)

326. He testified that the evaluation of fetal heart tracings is a dynamic process and he would commonly look back ten minutes and put it in the context of what has been happening for the previous four or so hours but going back to the previous day or more distant comparisons is not necessary. (TT Day 4, 21:12-22:18.)

327. In response to the question of whether tracings showing late decelerations in the early labor course "like the day before" mean that the fetus is suffering some kind of

cumulative effect, Dr. Pettker responded that it is "very common for there to be periodic changes in the fetal heart monitoring, like seeing late decelerations that resolve, and, as long as, subsequently, there's normal fetal heart rate tracing, that would not be a situation that there's a cumulative impact or adverse impact of that brief time period, that it resolved, and that the status would be normal, at the time I'm assessing, as long as the fetal heart rate monitoring is normal." (TT Day 4, 22:19-23:5.)

328. In answer to the question of how a fetus returns to baseline after some decelerations and whether fetal reserves are depleted each time there has been some kind of hypoxic event, Dr. Pettker provided the following explanation:

> fetal acid-based physiology and the delivery of oxygen and release of carbon dioxide that happens across the placenta [is] an extraordinarily dynamic and replenishing process. [D]uring labor, there might be periods of time where there might be a disruption in oxygen delivery or carbon dioxide release that occurs past the placenta, but if that disturbance is relieved, then the fetus, in its recuperation mechanisms, will return to a baseline state as long as the oxygen/carbon dioxide delivery disturbance is relieved.
>
> And it's really common for, in fact, probably, most pregnancies, that there might be a period of time where there is a disturbance like that. But the natural physiology and development of human pregnancy and the placenta and the acid-based physiology of the fetus allows fetuses to recover and for that not to be a chronic impact on the fetus.

(TT Day 4, 23:6 -24:4.)

329. In response to the question of whether you see more subtle decelerations as the baby gets more sick, Dr. Pettker stated that

> [d]ecelerations will get worse as a fetus becomes more sick. So things become more apparent on the fetal heart rate monitoring. And that's why a Category III

> is highly suggestive of a fetus that may be at risk, because . . . that shows a pattern that is so striking. . . . [I]t wouldn't be that things become more subtle on the fetal heart rate monitoring as the baby gets more sick.

(TT Day 4, 24:5-14.)

330. Specific to this case, Dr. Pettker testified that he would not use the term "subtle decelerations" used by Dr. Mauldin because it is not

> a term that's used in the literature, and I would disagree that the presence of subtle decelerations, whatever those might be, would be an indicator of a very sick fetus. I think it's when people are looking back at fetal heart rate tracings and trying to interpret them, based on the result of a fetus's outcome, we want to find things that might be wrong in the monitoring that might have been happening, and we will look with a magnifying glass at fetal heart rate tracings to try to find where the signals might have been in the electronic fetal monitoring. And that might be what's done there. Had there been a normal outcome, in this case, in looking at the tracing, it's not apparent to me that we would be calling what she might be calling subtle as late decelerations, I think there's a retrospective bias, when we have the outcome.

(TT Day 4, 25:19-26:8.)

331. Dr. Pettker testified that, overall, the fetal heart rate tracing is Category I and Category II and did not give the appearance that it required cesarean section any time before it was performed. (TT Day 4, 27:13-15.)

332. More specifically, he stated that there were some normal features of the fetal heart rate tracing in the hour or two prior to delivery, including accelerations and moderate variability that would be classified as a Category I fetal heart rate tracing, there were episodic periods that had late decelerations and variable decelerations, he saw minimal variability and late decelerations from time to time which indicated a need for heightened

awareness of the status of the fetus and the progress of labor but none indicated a requirement for delivery, he never saw absent variability, and, citing specific tracings as examples, he saw accelerations throughout the labor progress as well as in the last two hours to be a reassuring indicator of a normal acid-based and oxygenation status of the fetus during those times. (TT Day 4, 27:17-30:13.)

333. When asked about the relationship between accelerations in the cited fetal tracings and hypoxic injury or acidosis, Dr. Pettker responded "nearly 100 percent of the time, if you have the presence of accelerations, it is reassuring for ruling out hypoxia" which meant that an hour before delivery and even closer to delivery he was nearly 100 percent sure that this fetus did not have hypoxia or acidosis. (TT Day 4, 30:14-23.)

334. Dr. Pettker also provided examples of Category I strips which occurred on December 17th, findings which were the basis for his disagreement with Dr. Mauldin that tracings in this case were a continuous Category II. (TT Day 4, 31:6-32:20.)

335. Dr. Pettker attributed the lack of any notation in the record about a Category I tracing to the way the interpretation of the fetal heart rate monitoring is documented:

> we are documenting what is occurring at exactly the time we are documenting[,] [a]nd we are documenting that 10-minute period around the time of our documentation . . . so it can be periods of documentation didn't correspond with the periods of the fetal heart rate tracing that were Category I. That doesn't mean that the nurses didn't see or the physicians didn't see Category I fetal heart rate tracings, at all, in the course of labor, it just means, at the time of the documentation, that they weren't seeing that.

(TT Day 4, 33:10-19.)

336. When asked whether a substantial number of physicians would have interpreted any of the tracings in this case as displaying acidosis and/or hypoxic injury in the fetus during the labor process, Dr. Pettker responded that he thought "there were periods of late decelerations that might indicate that there would be hypoxia, but this would be intermittent and resolved, and that there was not hypoxia that led to tissue injury that would lead to harm or death." (TT Day 4, 36:19-37:2.)

337. He also stated that he did not believe that a substantial number of physicians would think that these fetal heart rate tracings would indicate hypoxia or acidosis that led to injury. (TT Day 4, 37:8-10.)

338. Regarding notations in the record that Mrs. Gharbi was tachycardic during labor, Dr. Pettker stated that is not contributory to fetal heart rate tracings. (TT Day 4, 37:11-17.)

339. Regarding Dr. Mauldin's testimony that there was a 14-minute deceleration at approximately 7:30 p.m. on December 17th, Dr. Pettker responded that

> [t]here are various notes about a 14-minute deceleration. There is a decrease in the fetal heart rate around that time, and there is a bradycardia around that time. The definition of bradycardia is it's a fetal heart rate below 110 that has lasted more than 10 minutes. But during that part of the tracing, there's a lot of periods of indeterminate tracing, where you're actually not tracing. So it's really hard to come up with a continuous temporality to the length of the bradycardia.

(TT Day 4, 38:6-18.)

340. Dr. Pettker reviewed specific tracings relevant to the 14-minute time period at issue, and, after specifically describing what he saw, he expressed his conclusion that the

tracings did not indicate that the fetus was injured by hypoxic injury or acidosis during the period. (TT Day 4, 41:5-9.)

341. Dr. Pettker described the period as bradycardia rather than a 14-minute deceleration because the latter is not used in the guidelines for interpretation of fetal heart rate monitoring, explaining that "[i]f you have a decrease in the fetal heart rate below 110 for a period of more than 10 minutes, then, that's a baseline change and you would call that bradycardia." (TT Day 4, 41:10-20.)

342. After stating that a bradycardia can be "very concerning," Dr. Pettker added that "would require going into the room, doing appropriate resuscitative efforts, maybe, I.V. fluids, oxygen, turning the mother, assessing where we are in labor." (TT Day 4, 41:21-42:2.)

343. When asked how he knew there was no injury to the fetus during this period of time, Dr. Pettker stated "[i]t's my opinion, within a reasonable degree of medical certainty, that the returning to baseline and the variability seen after the tracing reassures me that this was not an event that caused permanent harm or damage. If that were the case, it would be that I would see severe abnormalities in the fetal heart tracing, like an absence of variability, after something like this." (TT Day 4, 43:4-13.)

344. Interpreting the fetal heart rate tracings following the bradycardia (Ex. J-3.207), Dr. Pettker stated that the tracings returned to normal at 7:50 p.m. and there was moderate variability. (TT Day 4, 43:17-21.)

345. In the tracings for the 7:53 to 8:00 p.m. period (Ex. J-3.208), Dr. Pettker again interpreted moderate variability and a Category II fetal heart rate tracing, pointing out that the unique pattern toward the end of the period is generally an indicator that a fetal scalp electrode was being used, adding that he would interpret the next period (Ex. J-3.209) in the same way—a Category II fetal heart rate tracing with no late decelerations. (TT Day 4, 43:22-44:17.)

346. Upon being questioned by the Court about the significance of the absence of late decelerations, Dr. Pettker replied

> the physiology behind late decelerations would be uteroplacental insufficiency or a disruption in the ability for the mother or the placenta to deliver oxygen or release carbon dioxide from the fetus. So they are connected with the physiology behind hypoxia and acidosis. So the absence of late decelerations is helpful for me to say that it's unlikely that we have hypoxia or acidosis, during this time.

(TT Day 4, 45:16-24.)

347. Dr. Pettker saw minimal variability for the next period (Ex. J-3.210) and moderate variability throughout the following period (Ex. J-3.211) with a late deceleration which Dr. Pettker found "not not concerning" but not signifying a "fetus in peril" because he did not see recurrent late decelerations. (TT Day 4, 46:2-48:4.) During the next time period (Ex. J-3.212), he saw moderate variability with no late decelerations, a Category I fetal heart rate tracing, followed by tracings (Exs. J-3.213, J-3.214) with minimal variability and no late or variable decelerations, Category II tracings. (TT Day 4, 48:7-49:25.)

348. When asked if he saw late decelerations with over half of the contractions for

tracings beginning at about 8:54 p.m. through 9:11 p.m. (Exs. J-3.215, J-3, 216), Dr. Pettker responded that he did not because he would have to see over four or more late decelerations and he identified eight detectable contractions and two late decelerations during this time period. (TT Day 4, 49:1-13.)

349. Regarding the evaluation of the fetus at the time of delivery for possible low oxygen episodes that occurred during the labor process, Dr. Pettker stated that the most indicative measure of the oxygen status at the time of delivery would be umbilical cord blood gases. (TT Day 4, 49:14-21.)

350. He confirmed that limb contractures would not indicate anything about the oxygen supply to the fetus during labor and stated that he had not seen limb contractures at delivery in cases of hypoxic and acidotic injury. (TT Day 1, 49:22-50:5.)

351. Dr. Pettker testified that his opinion would not change based on the fact that limb contractures were not seen on prenatal ultrasounds because, although very severe limb contractures can be see prenatally, E.G."had subtle, . . . non-severe limb contractures, and the assessment of limb contractures can be very difficult . . . prior to delivery, in utero, on ultrasound, because the fetuses are rolled up in a ball." (TT Day 4, 50:7-19.)

352. Dr. Pettker provided information about his review of cord blood bas results in this case:

> If we're just to look at the values, themselves, the pH read as 7.353, the oxygen saturation was 57, the partial pressure of oxygen, which is the P02 was 31, the bicarbonate level was 21.5, that's the HC03, and then for neonatal cord blood gases, we use something called the base deficit, and that was 4. . . . These are

> normal cord blood gas levels on a venous cord blood gas. So the pH was normal, did not have acidosis, the oxygen levels were normal, as well, and a normal base deficit means that the respiratory and metabolic state of the baby was such that it wasn't an uncompensated acidosis, . . . there was no presence of acidosis.

(TT Day 4, 51:15-52:3.)

353. In response to the notation "abnormal" on the lab results, Dr. Pettker said that he does not pay attention to such notations because he looks at the values themselves and knows what is normal and what is not and, in this case, 7.353 is normal and base deficit of 4 is normal and the reference ranges that he uses in practice for a base deficit would be 12 and a pH would be less than 7.0. (TT Day 4, 52:4-15.)

354. When asked about the effect of a delay in drawing the blood sample, Dr. Pettker responded that, if there is a delay (which generally means more than an hour to obtain the cord blood gas with the cord just sitting on a table in the interim), the values become more abnormal rather than more normal so, if there was a significant delay in this case, the result would have tended toward the abnormal and not gone toward the normal range. (TT Day 4, 52:16-25.)

355. In comparison to Dr. Mauldin's belief that the reason the records don't show an abnormal pH is because there was no time for the blood gas to equilibrate, Dr. Pettker stated that

> [i]f here had been hours of hypoxia that contributed to injury, that would contribute to death, we would, most definitely, see an abnormal blood gas, because we would have to see a totally uncompensated abnormality that a fetus could not overcome with physiology, and that would, most definitely, show

> up on a blood gas, including a venous blood gas. [These lab results] do not indicate . . . that hypoxia and/or acidosis occurred that was present in labor.

(TT Day 4, 53:1-18.)

356. Regarding the testimony that there was insufficient time to see tissue damage on autopsy or evidence of brain damage despite alleged hypoxic injury and acidosis in utero, Dr. Pettker stated he believed that

> if there was hypoxia and acidosis that would lead to death in a baby like this, . . . we would have to see tissue injury that would have led to cause of death, that we would see in some organ, in the brain, the kidneys, the heart, something that lead to the death of the baby, due to tissue damage from acidosis or hypoxia.

(TT Day 4, 54:17-55:1.)

357. Dr. Pettker stated that hypoxic injury or acidosis does not cause the three conditions seen on autopsy—contractures, pyloric stenosis, and Hirschprung's condition (which he described as "the dilation of the bowel, the colon and abnormal neurons and neuronal development around the colon")--in that these are genetic conditions due to genetic and organ development and, based on the constellation of signs (also including polyhydramnios), he believed that, although they were not fatal developmental disorders, "the constellation of these things made me concerned and gave me a high degree of suspicion that there was a very rare developmental abnormality that would have led to a constellation of findings, and that would have led to death in a baby like this, at the time of birth." (TT Day 4, 55:2-56:10.)

358. He also stated that even if he did not have the information about the conditions

found on autopsy, his conclusions

> about hypoxia and acidosis, and leading to tissue injury, leading to death, I would have made my conclusion independently of these findings, but I think whenever we're trying to explain a sudden and unexpected death in a neonate, at the time of birth, we also want to find out what happened and why it might have happened. And it helps be to come up with an explanation, a most likely explanation for us and for the family, of what probable occurred.

(TT Day 4, 56:23-57:14.)

359. Dr. Pettker noted that, while it is a helpful for the medical provider and the parents to find explanations for what has happened, there are not always explanations. (TT Day 4, 58:2-19.)

360. Dr. Pettker opined that, to a reasonable degree of medical certainty, he did not think the outcome would have changed with an earlier delivery, even if the baby was delivered a day or two days earlier. (TT Day 4, 57:15-24.)

361. On cross-examination, in the course of an extensive discussion about polyhydramnios and whether Dr. Pettker considered normal ultrasounds following the finding of polyhydramnios, Dr. Pettker confirmed that he considered them but added that he considered the finding of polyhydramnios on an ultrasound performed by a Maternal Fetal Medicine ("MFM") physician in an accredited Institute for Ultrasound and Medicine ("AIUM") facility to be an important part of the record where he could not comment on the other ultrasounds that were not done by an MFM in an AIUM facility. (TT Day 4, 59:9-63:25.)

362. In response to questioning about the contrast between Dr. Pettker's finding that contractures and Hirschsprung's Disease were evidence of E.G.'s developmental problem

and the autopsy's conclusion of "possible" Hirschsprung's Disease and the assessment that the limb contractures in congenital syndromes associated with both of the abnormalities were "generally much more pronounced that seen in this baby's case," Dr. Pettker stated that he did not change his assessment because the first part of the autopsy report which is the final anatomic diagnosis did not include "possible" in relationship to Hirschsprung's Disease and he found the use of the word "generally" related to the degree of contractures to be "a relevant qualifier." (TT Day 4, 65:10-67:5.)

363. When asked to confirm that the pathologist pointed out that E.G.'s genetic tests he ordered were normal, Dr. Pettker clarified that the chromosome test was normal and agreed that the pathologist did not order any further genetic testing. (TT Day 4, 67:24-68:8.)

364. Dr. Pettker stated that, in his practice, when he sees a constellation of congenital abnormalities, he works with a geneticist to determine "way beyond karyotype" what an appropriate genetic workup would be. (TT Day 4, 68:8-11.)

365. Finally, Dr. Pettker confirmed that Hirschprung's Disease is treatable and children do not die of contractures, but he disagreed with Plaintiffs' counsel's follow-up statement that, "[i]f there's no polyhydramnios, that's not a constellation," responding that "I am not going to agree with you that there was not polyhydramnios in the pregnancy." (TT Day 4, 68:18-69:3.)

- **TONI GOLEN, M.D.**

366. Toni Golen, M.D., an Obstetrician and Gynecologist at Beth Israel Deaconess

Medical Center in Boston, Massachusetts, was offered by Defendant and admitted as an expert in the field of Obstetrics and Gynecology. (TT Day 5, 8:6-7, 10:5-7.)

367. Based on her review of the entire medical record, Dr. Golen opined to a reasonable degree of medical certainty that Dr. Francis met the standard of care in the management of Mrs. Gharbi's labor and delivery, including her evaluation of Mrs. Gharbi over the course of the induction of labor and labor, and performance of the C-section at approximately 1:41 a.m. on December 18, 2017. (TT Day 5, 10:9-23.)

368. Regarding sign out and when an attending physician first sees a patient in labor or is undergoing induction, Dr. Golen said she was not concerned that the medical records did not contain a full summary or narrative of the sign-out and she explained that it can be seven hours or more before an attending sees a woman in labor but the doctor is staying in touch with nurses and residents in the interim. (TT Day 5, 12:11-16:2.)

369. Regarding a plan of care for Mrs. Gharbi, Dr. Golen stated that that there were notes which indicated Dr. Francis's plan of care but that the notes had not been written by Dr. Francis. (TT Day 5 73:2-5.)

370. Dr. Golen confirmed that it would not always be necessary to review prenatal records or the written records and fetal heart tracings from the time before sign out but there may be instances where she would do so. (TT Day 5, 16:12-17:9.)

371. She opined that it would not fall below the standard of care if the attending does not look at strips from the day before because the fetal heart rate tracing is a real-time

measurement of the fetal heart rate--the attending is responsible for and wants to be knowledgeable about what is happening because decisions are made based on what is happening currently in terms of the status of the fetal heart rate tracing. (TT Day 5, 17:10-25.)

372. Regarding the stopping and starting of Pitocin, Dr. Golen explained that was normal and the administration of Pitocin/Oxytocin was the province of the nurse (sometimes together with the residents). (TT Day 5, 20:13-22:24.)

373. Dr. Golen opined that Dr. Francis did not fall below the standard of care in the amount of time she spent with Mrs. Gharbi, her reliance on residents and nurses, and her awareness of her patient's status, because the management of the labor and of Mrs. Gharbi was "entirely within the standard of care and there was no point at which there is a question of whether something did or did not happen at a time that it should have." (TT Day 5, 24:8-25:5.)

374. Dr. Golen testified that, when she sees decelerations, they must be assessed in the context of how frequently they are happening, the variability and the baseline. (TT Day 5, 29:9-14.)

375. She explained that recurrent decelerations can be normal and, in some situations, they use a process called intrauterine resuscitation which is aimed at increasing blood flow through the placenta and is accomplished by changing the patient's position, giving intravenous fluids, and sometimes oxygen. (TT Day 5, 29:16-30:5.)

376. Dr. Golen stated that she agreed with the way Dr. Francis interpreted fetal heart rate tracings during labor and whether they indicated a C-section in this case, Dr. Francis's interpretation was consistent with ACOG and NIH definitions, and she believed that a substantial number of OB-GYN physicians would also agree. (TT Day 5, 30:13-23.)

377. Dr. Golen testified that it made a difference whether you were interpreting the fetal heart rate tracings in real-time or after the fact (as she and Dr. Mauldin were) because the determination or conclusion about what a fetal heart rate tracing represents is influenced by the outcome when it's known so we know that retrospective review of fetal heart rate tracings is biased by the outcome. (TT Day 5, 30:24-31:7.)

378. Turning to a review of the fetal heart rate tracings in this case, Dr. Golen stated that the tracings are Category I and Category II throughout. (TT Day 5, 32:2-4.)

379. In response to the question of whether a continuous Category II strip, by itself, requires intervention, Dr. Golen said a continuous Category II strip is when intrauterine resuscitation is provided but there is not a specific action required other than that and C-section is not required. (TT Day 5, 5-14.)

380. Dr. Golen did not see any place in the fetal heart rate tracings where there was absent variability. (TT Day 5, 32:15-17.)

381. Regarding arrest of labor, Dr. Golen testified that Mrs. Gharbi had the same exam from 3 p.m. until 6.40 p.m. on December 17th but she did not have arrest of labor at 7:00 p.m.:

> The definition for arrest of labor is that the cervix does not change, at all, after six centimeters, for four hours, with ruptured membranes, in the face of adequate contractions. And she did not meet this criteria. We didn't know that her contractions were adequate, and there was not four hours that had gone by.

(TT Day 5, 33:1-17.)

382. Dr. Golen further testified that it did not fall below the standard of care to allow Mrs. Gharbi's labor to continue after 7:00 p.m. based on the fetal heart tracings and the progress of labor because she had a normal labor pattern for a first-time mother, she was just getting into the active labor phase and the standard of care, in this situation, would be to continue to monitor for contractions, cervical dilation and fetal heart rate tracing. (TT Day 5, 33:18-34:1.)

383. Dr. Golen specifically reviewed the time period beginning at 7:24 p.m.:

> starting at 7:24, in order to assign a baseline, I just need to look ahead a little bit. So the baseline is about 140 with some variable decelerations and . . . minimal variability and six contractions that I can see up until about 7:38 or so. There's a loss of contact or discontinuity, loss of fidelity in the tracing, so there's no recording of the heart rate starting at about 32, and then from 39 to 41 or so, it looks to me like that's a maternal heart rate that's being recorded, still loss of fidelity, and then a return of fidelity at, approximately, 47 or so, with a baseline, again, at that point, of about 120.[5]

(TT Day 5, 34:10-21.)

384. When asked whether there was a 14-minute deceleration during the time

[5] Dr. Golen's reference to a "baseline" of "120" and "140" refers to heartbeats per minute ("BPM"). As set out in the Glossary section of this Memorandum Opinion, a normal heart-rate pattern for a fetus would be a baseline of the heart rate between 110 and 160 beats per minute. *See supra* p.10.

period she reviewed, Dr. Golen stated that there was not a 14-minute deceleration on the monitor and there was no bradycardia on the monitor. (TT Day 5, 34:22-35:1.)

385. Explaining her conclusions, Dr. Golen said there was not a signal and during a time when there is no signal, which is "very, very, common during labor," there's not a fetal heart rate tracing during that time on the monitor. (TT Day 5, 35:3-14.)

386. When asked if she had ever seen a 14-minute deceleration, Dr. Golen said that a 14-minute deceleration is not an accurate description of an event on a fetal heart rate tracing, it is not an occurrence that happens—it is either a deceleration or a change in baseline. (TT Day 5, 82:19-25.)

387. Dr. Golen disagreed with Dr. Mauldin that this period on the fetal heart tracings is a Category III tracing. (TT Day 5, 36:5-9.)

388. Regarding the record report of a 14-minute deceleration, Dr. Golen said that the notation did not change her opinion that there was no 14-minute deceleration because the source of the truth is in the fetal heart rate tracing and the "particular note was written a long time after that had happened, and was the report from one person to another in the context of CPR going on for the baby." (TT Day 5, 35:15-36:1.)

389. As to how the fetus's status should be assessed during a time when the monitor is disconnected, Dr. Golen said you need to gather information from before the monitor disconnected and after it is reconnected to give you information about what might have happened during the period when you didn't have contact, adding that this "happens

all the time" and "is an extraordinarily common occurrence during labor." (TT Day 5, 36:10-25.)

390. Dr. Golen confirmed that a C-section at 8:00 p.m. on December 17, 2017, was not called for based on what is seen during the period of lost tracings and before and after. (TT Day 5, 37:1-5.)

391. For the period after which contact was reestablished, Dr. Golen found that the heart rate returned to normal and variability was moderate in some places and minimal in others, implying that there was not fetal acidemia at that point. (TT Day 5, 37:22-25.)

392. Looking at the fetal heart tracings just before 1:00 a.m. on December 18, 2017, Dr. Golen saw a baseline of 120 at 12:56, an acceleration at approximately 1:00, an acceleration at 1:10 and moderate variability. (TT Day 5, 38:12-39:5.)

393. Dr. Golen stated that an acceleration is "a reliable predictor that there's not acidemia, it's the one . . . isolated characteristic of the fetal heart rate tracing that gives us reliable information that there's not acidemia" and what she saw in the period from just before 1:00 to 1:10 was "all indicative of a healthy acid-based balance and not an acidemic or hypoxic fetus." (TT Day 5, 38:15-21, 39:5-7.)

394. Dr. Golen did not agree that decelerations get more subtle as the fetus gets sicker. (TT Day 5, 39:8-11.)

395. Based on her review of the fetal heart tracings, Dr. Golen concluded that they did not contain any evidence that the fetus was injured during the period of disconnection

starting at around 7:24 p.m. on December 17th and the tracings did not contain any indication that the fetus was injured by low oxygen from then until the monitor was disconnected before the C-section. (TT Day 5, 39:21-40:7.)

396. Dr. Golen opined that a C-section was not called for at any time before Dr. Francis performed it based on the fetal heart rate tracings and the progress of labor. (TT Day 5, 40:8-11.)

397. She also opined that Dr. Francis did not fall below the standard of care by not having specifically discussed a C-section with Mrs. Gharbi until she did on the morning of December 18, 2017. (TT Day 5, 84:5-17.)

398. Dr. Golen confirmed that entries found in hospital records from 9:30 p.m. on December 17, 2017, to 1:00 a.m. on December 18, 2017, regarding fetal heart rate tracings, the administration of supplemental oxygen, and Mrs. Gharbi's tachycardia and tachysystole did not change her opinion. (TT Day 5, 40:19-43:14.)

399. Regarding the need to disconnect the fetal heart rate monitor pre-surgery and the evaluation of the fetus during that time, Dr. Golen explained that you look at what immediately preceded disconnecting the monitor and do a spot check on what the heart rate is. (TT Day 5, 43:22-44:20.)

400. In this case, the fetal heart tracing preceding discontinuation was normal and the spot check showed a heart rate of 120 beats per minute. (TT Day 5, 44:13-15, 45:1-14.)

401. Dr. Golen stated that the outcome in this case was not predictable and

confirmed that E.G.'s survival after delivery was not affected by the management of the labor process. (TT Day 5, 46:17-25.)

402. Dr. Golen explained that resuscitation after delivery, particularly after a cesarean delivery, is very common and babies don't necessarily go through the same transition process as they would with a vaginal delivery. (TT Day 5, 47:4-12.)

403. When asked whether there was any way to confirm that the fetal heart tracings did not indicate hypoxic injury or acidosis in the fetus during labor, Dr. Golen said it could not be confirmed until the baby was born but once the baby is born, taking a cord blood sample for gas analysis (cord blood gas analysis) tells whether the baby was acidemic when the baby was still inside the uterus and, in this case, they retrieved a cord blood gas sample and it was normal. (TT Day 5, 47:19-48:3.)

404. Dr. Golen was asked whether a physician can diagnose hypoxic injury and/or acidosis of the fetus from fetal heart rate tracings and responded that it could not be diagnosed because there was no objective measure of low oxygen of the fetus in utero. (TT Day 5, 31:8-13.) Dr. Golen further explained that electronic fetal monitoring is not a diagnostic tool that is used to assess for fetal hypoxia or acidemia in utero but rather is a screening tool where the doctor looks for things like decelerations and minimum or absent variability. (TT Day 5, 50:1-24.)

405. In sum, Dr. Golen opined that Dr. Francis met the standard of care in this case with regard to the decision-making for the timing and mode of delivery of E.G. (TT Day 5,

48:13-7.)

406. Regarding her experience with a baby dying right after delivery, Dr. Golen confirmed that she had experienced this type of situation and said that

> [o]ne of the most difficult parts of [the experience] is that we often don't have an explanation. And it seems like we should be able to, we have got all sorts of medical advances and fancy tests that we can do, and it seems logical we ought to be able to come up with an exact reason. Unfortunately, in Obstetrics, that's not the case. And more often than not, when tragedies like this happen, we actually don't know what the reason was.

(TT Day 5, 48:18-49:8.)

407. Dr. Golen confirmed that the neonatologist recorded that she was asked to come to the delivery "because of fetal distress with symptoms of variable decelerations." (TT Day 5, 56:19-23.)

408. When asked on cross-examination to confirm that she did not believe there was a 14-minute deceleration or that the baby had fetal distress according to the records, Dr. Golen reiterated that "there wasn't a 14-minute deceleration, and fetal distress is not something that has a definition." (TT Day 5, 56:24-57:3.)

409. In a discussion about polyhydramnios during Mrs. Gharbi's pregnancy, including the fact that there were normal ultrasounds before and after the November ultrasound showing polyhydramnios, Dr. Golen stated that there was a difference between ultrasounds done by a Maternal Fetal Medicine specialist and the others which Plaintiffs' counsel referenced, the former being "official ultrasounds" and the latter being done at the bedside with a portable machine, but, either way, polyhydramnios was documented and the

fact that it was there at one point is not arguable. (TT Day 5, 59:7-64:14.)

410. Concerning arrest of labor, Dr. Golen disagreed that Mrs. Gharbi experienced arrest of labor on December 17th in that the time between exams (3:00 p.m. and 6:42 p.m.) was not four hours and there was no assessment of the adequacy of contractions so the situation did not meet the definition of arrest of labor. (TT Day 5, 69:16-70:21.)

411. On further questioning about whether there was arrest of labor at 7:00 p.m. when the dilation had not changed from 7 centimeters since 3:00 p.m., Dr. Golen stated that there was not arrest of labor at that point because, although still 7 centimeters, "the effacement has changed, and it's zero station, so that's a change, and, again, we don't know the adequacy of contractions" and a notation of regular contractions is not adequacy. (TT Day 5, 72:1-10.)

412. In response to questions about the lack of notes by Dr. Francis and her lack of awareness that the resident was going to restart Pitocin on December 17th, Dr. Golen stated that it was acceptable and common for residents to make decisions together with the nurse about starting and stopping Pitocin at various points during labor and the attending may not know about it in real time. (TT Day 5, 74:22-75:4.)

413. Turning to the venous cord blood sample and the fact that some values were flagged as abnormal in the record showing the results of the sample drawn from the umbilical cord, Dr. Golen explained (1) the normal values indicated on the form were normal values for adults but not for samples drawn from umbilical cords as in this case, (2) different

values apply for analyzing fetal circulation which was being tested with the umbilical cord sample here, and (3) because the values identified on the form did not apply to fetal circulation, the values flagged on the lab result form as abnormal were not relevant in this case. (TT Day 5, 75:9-76:11.)

414. Regarding the effect of a delay in sampling, Dr. Golen disagreed that the results would necessarily be invalidated and it would depend on the period of delay but, because the pH changes over time and decreases over time before it's collected and processed, the pH indicated here is certainly valid and added that, in fact, the pH may reflect something slightly lower that it would have been if it were half an hour earlier. (TT Day 5, 76:12-25.)

415. When asked to confirm that billing code ICD-10-P84, which was listed as the final diagnosis for E.G. (Ex. J-4.1), included the terms "[a]cidemia of the newborn, acidosis of the newborn, anoxia of the newborn, asphyxia of the newborn, hypercapnia of the newborn, hypoxemia of the newborn, hypoxia of the newborn, and mixed metabolic and respiratory acidosis of the newborn," Dr. Golen said she was not familiar with the code as recited by counsel and the code was identified by a non-medical person as part of an administrative coding process that happens in a billing office to send bills to the insurance company and has nothing to do with patient care. (TT Day 5, 79:2—80:17.)

416. Dr. Golen confirmed that the ICD-10 code did not change her opinion that the fetus did not have hypoxic injury and/or acidosis at delivery. (TT Day 5, 85:16-19.)

417. When asked by the Court to describe, in narrative form, why the standard of care was not breached in this case, Dr. Golen responded as follows:

> When Obstetricians embark on an induction and manage labor, we have a set of responsibilities, and in order to carry out those responsibilities, we rely on certain pieces of data. This isn't exclusive, but some important pieces of data are the physical exam, including dilation of the cervix, and that informs us as to whether cervical or labor progress is normal.
>
> So that's one . . . block of our reasoning, is labor progressing normally? Is physiology proceeding as it ought to? Are we doing a good job carrying out this induction in trying to imitate the natural process? So that's one piece.
>
> The other piece is, as best we can tell, are the mom and baby healthy during the process? With regard to mom, we have a multitude of pieces of data that we can rely on, blood tests and imaging and physical exams to tell us that, so it's quite reliable, it's quite diagnostic.
>
> With regards to the fetus, our ability and the instruments that are available to us are extraordinarily limited and, really are limited to one test only, which is the external, in most cases, fetal heart rate monitor, in some cases, it's an internal monitor, in this case, it was both. That is an extraordinarily rudimentary test that actually performs not very well, in terms of predicting outcomes, and we know this, this has been proven time and time again since the early 70's when it came into being and was used.
>
> It is commonly used, that's indisputable, but there's a variety of reasons why it's commonly used. It's not commonly used, though, because it is highly predictive of outcomes, it is commonly used because it has become a culture of that's what we do.
>
> The test is a screening test, and screening tests are good at picking up some things but not good at picking up others. This particular screening test, the fetal heart rate monitor, is very good at detecting when babies have normal acid-based balance, we call it Category I fetal heart rate tracing. And it's pretty good at definitively saying when there's not normal acid-based balance, which we call Category III fetal heart rate tracing.
>
> But this is problematic, because the vast majority of babies are in

> Category II, at some point or another, or even the majority of the time that they're in labor. So this leaves us to need to combing these two buckets of reasoning. Is labor progressing normally? Is my best judgment that this is a normal fetus with a normal acid-based balance, and can we continue?
>
> This is what we do, as Obstetricians, every moment of every day. We combine these two things together, and we decide, moment by moment, Do we still go ahead? And in this case, there was no evidence that labor had stalled. I am absolutely firm in the fact that the patient never met the criteria for an arrest of dilation, by any standard, and there was never any indication that this baby had abnormal acid-based balance.
>
> So those two pieces coming together, the standard of care would dictate to continue to monitor and proceed with the induction until there was clear evidence that it wasn't working, when it did become evident, in the second stage of labor, and she wasn't delivering the baby after pushing.
>
> So inasmuch as there's a lot of technical details and wording and through jargon around this, obstetrical management really does rest on these two very important principles, and in this way, this patient was managed absolutely within the standard of care.

(TT Day 5, 87:10-89:22.)

- **RICHARD POLIN, M.D.**

418. Dr. Polin, a professor of Pediatrics at New York Presbyterian Hospital, the hospital of Columbia University, whose clinical practice is intensive care for newborn babies, was offered by Defendant and admitted as an expert in the field of Neonatology and Pediatrics. (TT Day 6, 3:22-4:4, 7:3-4, 8:24-25.)

419. Dr. Polin opined to a reasonable degree of medical certainty that E.G. did not die of hypoxia or acidosis during labor based on the data from the umbilical venous cord blood specimen which did not demonstrate acidemia. (TT Day 6, 9:16-10:3.)

420. He later confirmed that, although the chart noted prolonged decelerations and decreased variability during labor, his opinion was based purely on the objective evidence of normal acid-based balance in the blood. (TT Day 6, 23:25-24:8.)

421. Regarding the lack of an arterial sample, Dr. Polin stated that if an arterial sample had been obtained, it would likely have reflected similar values to the venous sample. (TT Day 6, 25:22-24.)

422. When asked how he interpreted "abnormal" on the cord blood gas results, he said he assumed that the hospital labeled the results as abnormal based on the standards they set for cord blood measurement and the ranges identified signified what the lab had decided are normal ranges but they are not necessarily national standards, just the hospital's own standards for that specimen. (TT Day 6, 11:17-12:5.)

423. Dr. Polin added that the ranges did not impact his interpretation of the objective data points identified in the results. (TT Day 6, 12:6-8.)

424. Regarding resuscitation, Dr. Polin stated that many babies require some measure of resuscitation and it is abnormal for a baby not to be able to be resuscitated. (TT Day 6, 12:20-24.)

425. Dr. Polin stated that hypoxic injury and/or acidosis developed before birth did not cause limb contractures that were visible on delivery in this case. (TT Day 6, 12:25-13:3.)

426. On cross-examination, Dr. Polin confirmed that he knew Dr. Chung, she had

written chapters for books he has written, and he had not collaborated with her in reviewing medical/legal cases in general or in this case, and they did not work together in preparing their reports and Declarations in this case. (TT Day 6, 13:21-14:25.)

427. After a comparison of his Declaration and that of Dr. Chung and being questioned about the preparation of his Declaration, Dr. Polin stated that he may have talked with Ms. Parker, but it was his language and likenesses between the two were coincidental. (TT Day 6, 15:22-17:22.)

428. Regarding the indication in his report that Mrs. Gharbi's prenatal course was remarkable for polyhydramnios, Dr. Polin disagreed with Plaintiffs' counsel that there was never a diagnosis of polyhydramnios, stating that the chart indicated that there was a concern about upper normal and moderate polyhydramnios. (TT Day 6, 20:5-23:5.)

429. When questioned about the pathologist's findings regarding Hirschsprung's Disease, contractures, and E.G.'s skin and lungs, Dr. Polin recognized discrepancies between his report and the pathologist's findings and focused on the unusualness or abnormality of the findings both made. (TT Day 6, 27:9-34:7.)

430. Dr. Polin was asked to agree that "upon delivery, a baby that does not have a heartbeat, that doesn't have respirations, that doesn't cry, that is pale, has non-responsive pupils, is hypothermic and has non-responsiveness to resuscitation fits the criteria of being dead," and answered that "[i]f there is no response to resuscitation after twenty minutes, the baby is dead, but to say the baby is dead, when they obtained a heart rate in this case, the

baby is not dead, they were unable to perform a successful resuscitation unfortunately." (TT Day 6, 34:23-35:7.)

431. Regarding normal results of the testing done in this case, Dr. Polin testified that the testing done was only chromosomal studies which give limited information about genetic disease and the only way to look for genetic disease is to do whole exome genome sequencing which was not done in this case. (TT Day 6, 35:13-18.)

432. Dr. Polin did not know why further testing was not ordered but he said, in his hospital, the baby definitely would have had at least whole exome sequencing and probably whole genome sequencing. (TT Day 6, 35:19-25.)

433. Dr. Polin confirmed that he arrived at his own opinions and did not necessarily adopt the pathologist's opinions based on what he saw in all the records in the case. (TT Day 6, 38:13-17.)

434. When asked by the Court to explain the difference between an arterial blood gas result and a venous result related to hypoxia and acidosis, Dr. Polin stated that the arterial specimen would give slightly different pieces of information, but the pH measurements would both reflect acidemia or acidosis. (TT Day 6, 38:8-24.)

435. Dr. Polin then confirmed that the absence of an arterial specimen did not in any way invalidate the opinion he had given based on the venous blood gas results. (TT Day 6, 38:25-39:4.)

436. Dr. Polin also confirmed that a delay in collecting or testing the specimen would

result in the pH going down so, even if the sample had sat around, the pH value that is normal and does not indicate acidosis. (TT Day 6, 41:19-43:7.)

437. When asked by the Court to explain the significance of the pH value going up or down, Dr. Polin stated that

> [i]f the baby is experiencing hypoxia and lack of oxygen delivery to tissues, the response in all of us, in a fetus or newborn, it can make certain acids, which accumulate in the blood, and those acids, like lactic acid, . . . actually cause the pH to fall. The more the acid accumulation, the lower the pH. So if I see an absence of acidemia, it suggests that the baby did not have prolonged hypoxia causing production of those acids prior to birth. In every baby I've ever seen with prolonged hypoxia, there's . . . always an accumulation of lactic acid in a falling pH.

(TT Day 6, 43:24-44:11.)

438. Dr. Polin also confirmed that an abnormal pH indicating acidosis would be below the 7.353 recorded here. (TT Day 6, 44:15-17.)

439. He further noted that he did not use the reference ranges provided on the report and the values were probably consistent with what's found in the venous blood of an adult as opined by Dr. Pettker. (TT Day 6, 44:20-45:7.)

III. CONCLUSIONS OF LAW AS TO LIABILITY

1. Plaintiff brings this action for medical malpractice under the Federal Tort Claims Act ("FTCA"), 28 U.S.C. §§ 2671, et seq., because Dr. Francis was employed by a federally funded health clinic at the relevant time. (*See* Doc. 1 ¶ 1.) The FTCA "provides a mechanism for bringing a state law tort action against the federal government in federal court," and the "extent of the United States' liability under the FTCA is generally

determined by reference to state law." *In re Orthopedic Bone Screw Prod. Liab. Litig.*, 264 F.3d 344, 362 (3d Cir. 2001) (quoting *Molzof v. United States, 502* U.S. 301, 305 (1992)).

2. The law of Pennsylvania is applicable in this case.

3. Under Pennsylvania law, "when a plaintiff's medical malpractice claim sounds in negligence, the elements of the plaintiff's case are the same as those in ordinary negligence actions." *Toogood v. Rogal*, 824 A.2d 1140, 1145 (Pa. 2003); *see also Merlini ex rel. Merlini v. Gallitzin Water Auth.*, 980 A.2d 502, 506 (Pa. 2009).

4. Under Pennsylvania law,

> [f]or a party to prevail in a negligence action, ordinary or professional, the elements are identical: the plaintiff must establish [1] the defendant owed a duty of care to the plaintiff, [2] that duty was breached, [3] the breach resulted in the plaintiff's injury, and [4] the plaintiff suffered an actual loss or damages.

Merlini 980 A.2d at 506 (citing *Martin v. Evers*, 711 A.2d 458, 461 (Pa. 1998)).

5. "[M]edical malpractice can be broadly defined as the 'unwarranted departure from generally accepted standards of medical practice resulting in injury to a patient, including all liability-producing conduct arising from the rendition of professional medical services.'" *Merlini*, 980 A.2d at 506 (quoting *Toogood*, 824 A.2d at 1145). Proof of medical malpractice in Pennsylvania requires evidence of the standard of medical care and a showing by a preponderance of the evidence that there was a departure from this standard. *Vlases v. Montgomery Ward*, 377 F.2d 846, 851 (3d Cir. 1967) (Pennsylvania law requires a preponderance of evidence in favor of plaintiff's basic proposition, but the

trier of fact need not be persuaded beyond a reasonable doubt). "Because the negligence of a physician encompasses matters not within the ordinary knowledge and experience of laypersons a medical malpractice plaintiff must present expert testimony to establish the applicable standard of care, the deviation from that standard, causation and the extent of the injury."[6] *Toogood*, 824 A.2d at 1145 (citing *Hightower-Warren v. Silk*, 698 A.2d 52, 54 (Pa. 1997)).

6. In a medical malpractice action, the duty requirement means that "[a] physician owes his patient a duty to employ that degree of knowledge, skill, and care ordinarily possessed by members of the medical profession." *Toogood*, 824 A.2d at 1150 (citing *Hodgson v. Bigelow,* 7 A.2d 338 (Pa. 1939)). This duty is not a requirement that the physician "be infallible, and making a mistake is not negligence as a matter of law. In order to hold a physician liable, the burden is upon the plaintiff to show that the physician failed to employ the requisite degree of care and skill." *Id.* (citing *Brannan v. Lankenau Hosp.*, 417 A.2d 196 (Pa. 1980); *Bierstein v. Whitman*, 62 A.2d 843 (Pa. 1949)).

7. The duty requirement is not at issue in this case. The parties dispute whether Dr. Francis breached her duty of care, whether the breach caused E.G.'s death, and what damages resulted.

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[6] *Toogood* goes on to explain an exception to the need for expert testimony which is not applicable in this case: "A very narrow exception to the requirement of expert testimony in medical malpractice actions applies 'where the matter is so simple or the lack of skill or care so obvious as to be within the range of experience and comprehension of even non-professional persons,' also conceptualized as the doctrine of *res ipsa loquitur.*" 824 A.2d at 1145 (quoting *Hightower–Warren,* 698 A.2d at 54 n.1.